THE

AMERICAN REVIEW OF TUBERCULOSIS
ABSTRACTS OF TUBERCULOSIS

VOLUME XXXIX MAY, 1939 ABST. NO. 4

CONTENTS

PAGES

Tuberculosis of Lungs (*concluded*) 61–63
Tuberculosis of Organs Other than Lungs 63–76
Pathology
a. *Anatomical* 76–80
b. *Physiological* 81–86
c. *Experimental* 86–89

Diabetes and Tuberculosis.—That the diabetic is susceptible to pulmonary tuberculosis, and that this susceptibility has increased since the use of insulin are well known. During a six year period, beginning January, 1932, there were admitted to the Waldhaus Charlottenburg Tuberculosis Hospital of Berlin 125 diabetics with tuberculosis. This constituted 1.7 per cent of the total admissions. The patients came from the lower middle class population and were mainly in the 45 to 50 year age group. At the time of this report 37 patients were still living and autopsies had been performed on 39 of the patients who had died. In four-fifths of the cases diabetes had preceded tuberculosis, the mild diabetic being less susceptible than the poorly managed moderately severe diabetic, and the average duration of the preëxisting diabetes being eighteen months. Where tuberculosis was the initial disease, in most instances, diabetes developed in six months and the course of the combined disease was more rapid and fulminating. Once the combination of both diseases, however, had occurred, the patients were subject to the complications of both diabetes and tuberculosis, but tuberculosis, rather than the severity of the diabetes, was the cause of death. Four-fifths of the patients were severe diabetics and all required insulin. Insulin susceptibility was more frequent than insulin resistance. Three-fourths of the patients were obese endocrine disturbance types. The exudative form of pulmonary tuberculosis was found in two-thirds of the cases, and the author was unable to observe the so-called "diabetic tuberculosis," either clinically or roentgenologically. The impression, however, that haematogenous tuberculosis is less common in diabetics was confirmed. With caution, the usual collapse measures can be employed as in nondiabetic tuberculosis. Laryngeal tuberculosis was present in 14 autopsied cases. A definite decrease in the incidence of tuberculous ulceration of the intestines was noted. While it has been stated frequently in the past that up to 90 per cent of diabetics with tuberculosis have intestinal involvement, in a series of 46 autopsies, 52 per cent had no evidence of intestinal ulceration. It is the opinion of the author that this decrease is unrelated to the influence of insulin, but is due to the exudative character of the tuberculosis, and more especially to the disturbed diabetic metabolism, with an additional factor of the high vitamin content of the diet in these cases.—*Über die Beziehungen des Diabetes mellitus zur Lungen- und insonderheit zur Darmtuberkulose, E. M. Müller, Ztschr. f. Tuberk., August, 1938, lxxx, 281.*—(*A. B. C.*)

Diabetes and Tuberculosis.—For the purpose of determining the incidence of pulmonary tuberculosis in diabetics living in London, 230 consecutive diabetic admissions were examined and X-rayed. Fifteen patients or 6.5 per cent were found to have pulmonary tuberculosis, but with the exception of 2 patients with active, extensive

pulmonary lesions, none presented abnormal physical signs. The X-ray appearance varied from early infiltrations to pneumonic consolidations. All 15 cases were untreated diabetics, and with subsequent adequate control the pulmonary lesions progressed favorably. A high caloric diet with a high carbohydrate and low fat content and the use of insulin are recommended. While specific collapse measures are indicated when pulmonary tuberculosis is associated with diabetes, the treatment of the diabetes should be the first consideration. The prognosis in these cases is excellent if the pulmonary lesions are discovered early, and for this reason routine chest X-ray films are advised for every diabetic, particularly in poorly controlled cases.—*Pulmonary Tuberculosis Complicating Diabetes Mellitus, H. P. Himsworth, Quart. J. Med., July, 1938, vii, 373.*—(*A. B. C.*)

Bovine Infection.—In a series of 118 cases of pulmonary tuberculosis, 2 were found to be due to bovine infection. Both patients were farmers. In cultures of the sputum bovine tubercle bacilli were isolated. Animal inoculations confirmed the cultural findings.—*Beitrag zur Frage der Bedeutung der bovinen Infektion bei Lungentuberkulose und der Möglichkeit der Züchtung des bovinen Tuberkelbazillus, M. Nüss, Ztschr. f. Tuberk., August, 1938, lxxx, 306.*—(*A. B. C.*)

Heart in Pulmonary Tuberculosis.—The report is based upon the study of 416 cases of active tuberculosis and of 44 inactive cases. Of the active cases, 38 had arterial hypertension and 14 of these had generalized arteriosclerosis. Mediastinal displacement is not an uncommon occurrence in pulmonary tuberculosis; this shifting of the anatomical position of the heart is a possible cause of the abnormalities of the QRS group of the electrocardiogram. In the 108 cases showing shift of the heart to the left, 26.8 per cent showed right axis deviation in the electrocardiogram, 9.2 per cent left axis deviation, and 64 per cent none at all. In the 44 cases showing shift of the heart to the right, 11.4 per cent showed left axis deviation, 13.6 per cent right axis deviation, and 75 per cent none at all. In 226 cases in which the heart was in normal position, 5.8 per cent showed left axis deviation, 12.4 per cent right axis deviation, and 81.8 per cent none at all. In 189, or 45 per cent, of the active cases, there were significant changes in the QRS complex, the RST segment, or the T-wave in the electrocardiogram. None of the inactive cases showed any important abnormality of the electrocardiogram. In 12 cases the Q wave was abnormal in that the Q_3 measured 25 per cent or more of the largest amplitude of the QRS in any one lead, a condition recognized by Pardee as being indicative of coronary disease. Thirty cases showed intraventricular block, 43 abnormality of the RST segment, and 119 abnormal T-waves. It is suggested that nutritional disturbance of the myocardium as a result of pulmonary tuberculosis of long standing may produce electrocardiographic changes similar to those found in coronary disease.—*The Heart in Pulmonary Tuberculosis; Electrocardiographic Consideration, W. R. Leverton, Ann. Int. Med., September, 1938, xii, 285.*—(*A. A. E.*)

Stenosis of Large Bronchi in Pulmonary Tuberculosis.—Eleven cases of bronchostenosis in pulmonary tuberculosis were reported in adults, the majority of whom were women. They were diagnosed on the basis of physical and roentgenological findings and then usually by bronchographic and bronchoscopic findings. Stenosing processes in the bronchus are very difficult to heal, and through valve formation produce various changes in the function and structure of the lung. The expiratory stenosis seems destined to defeat the effect of a technically harmless collapse therapy, owing to the emphysematous effect. In cases where a bronchiectasis has formed after the stenosis, collapse treatment can be dangerous because the discharge of a purulent secretion is stopped. There seems to be no safe treatment at present and the prognosis in advanced cases is doubtful.—*Einige Fälle von Stenosen der grösseren Bronchien bei Lungentuberkulose, E. Sandler, Acta tuberc. Scandinav., 1938, xii, 1.*—(*F. B. S.*)

Spontaneous Fractures of Ribs in Pulmonary Tuberculosis.—Among 10,304 radiographies of 4,570 cases of pulmonary tuberculosis the author has found 19 fractures of ribs, that is, 0.415 per cent. The fractures occurred almost always in the higher age groups in patients suffering from grave forms of pulmonary tuberculosis and twice as frequently in the female sex. As there was no history of trauma the author attributes the rib fractures to decalcification of the bones due to insufficient fixation of calcium in the tuberculous patient. This condition of osteoporotic bone atrophy results from a disbalance between apposition and absorption of calcium. The fracture, while usually spontaneous, is sometimes caused by sudden muscular contraction or twisting of the chest cage and causes a fairly constant pain which is intensified by coughing, sneezing and pressure. The frac-

ture heals usually in a regular way with callus formation.—*Studio radiologico delle fratture spontanee delle coste in sogetti affetti da tuberculosi polmonare, C. Sabbione, La Radiologia med., June, 1938, xxv, 528.*—(*F. G. K.*)

Pregnancy and Tuberculosis.—A study of the effect of pregnancy on the course of tuberculosis has been estimated from three types of data available at Olive View Sanatorium, namely, the extent of tuberculosis found at admission in patients with various pregnancy histories, the response to treatment of patients who became pregnant while in the Sanatorium, and the effect on the future life expectancy of those who became pregnant after leaving the institution. The records of 2,633 married women were examined, of whom 500 had never become pregnant according to their histories and 718 had never borne a living child. Of the women with a history of pregnancy, more than a third reported only one child, and the average number of children per woman was only 2.2, although many more pregnancies were recorded. The average length of time at admission since the last pregnancy had occurred was about five years. Since the average duration of married life was only nine years, with 2.4 pregnancies per woman, the average interval between a pregnancy and any chance event would have been even less. The pregnancies were therefore not significantly related to the incidence of the tuberculosis. The authors conclude that the tuberculous woman who becomes pregnant has a course not greatly different, so far as her tuberculosis is concerned, than non-pregnant patients, while so far as her pregnancy is concerned she does not greatly differ from other pregnant women. In cases of well healed, arrested tuberculosis many exertions may be safely undertaken that should be forbidden during a more active phase. In unstable conditions, when the future course of the disease hangs in the balance, pregnancy should be at least postponed. Contraceptive measures may sometimes be recommended to a woman with tuberculosis in whom, if pregnancy does occur, it may be allowed to proceed without interruption. With extensive advancing lesions and with greatly diminished respiratory reserve, on the other hand, every precaution must be taken to avoid overtaxing the patient's strength.—*Pregnancy and Tuberculosis, Jane Skillen & E. Bogen, J. A. M. A., September 24, 1938, cxi, 1153.*—(*G. L. L.*)

Readmissions to a Tuberculosis Hospital.—In 1936 there were 2,004 admissions to Sea View Hospital and of these 280 were readmissions (13.9 per cent). These readmitted patients were studied in respect to their previous condition and interval actions. It is apparent that the longer a patient remains in the hospital during his first admission, the less is the likelihood for readmission. The great majority of these recidivists had symptoms at time of discharge although the sputum may have been continuously negative for several months. These discharged patients may be classified in four groups: (A) patients discharged by the Medical Board (33.5 per cent), (B) those who signed out against advice (42.5 per cent), (C) patients sent to other hospitals (19.2 per cent), (D) those discharged for disciplinary reasons (4.6 per cent). The patients in group A left the hospital in relatively good condition and therefore few were readmitted during the first six months after discharge. However, there was a gradual increase in the number of readmissions from this group up to two years after discharge; this indicates the necessity for prolonged careful attention after discharge. In group B 50.5 per cent returned to the hospital within the first year after discharge; all of the recidivists in this group were readmitted for causes that may well have been eliminated by prolonged care at the time of the first admission and proper after-care. It is apparent that roentgen, sputum and physical examinations are all too infrequently done away from the hospital. A standardized procedure for the examination of sputa is advocated in order to make the interpretation of reports universally possible.—*A Study of All Readmissions to Sea View Hospital during 1936, I. D. Bobrowitz & J. L. Leon, Quart. Bull. Sea View Hosp., April, 1938, iii, 280.*—(*A. A. E.*)

Tuberculous Bronchitis.—Tuberculosis of the tracheobronchial tree is not infrequent and may appear in the following forms: (*1*) as a granulomatous mass, (*2*) as a superficial ulcer, (*3*) as a caseous necrotic process, and (*4*) as stenosis due to submucosal disease with an intact mucous membrane. In the ulcerative and caseous forms there may be tubercle bacilli in the sputum despite roentgenological signs of controlled disease in the lungs. Involvement of large branches may cause extensive atelectasis; or, if the obstruction is of the ball-valve type, obstructive emphysema may ensue. Eight cases, all in females, are reported; in all of these there was definite bronchial tuberculosis with little or no apparent pulmonary tuberculosis. The invasion of the bronchi was probably from adjacent tuberculous lymph nodes. Collapse therapy,

generally pneumothorax, has been thought the treatment of choice. This has been chosen in order: (*1*) to diminish the flow of air through the bronchus and thereby rest it; (*2*) to prevent aspiration and implantation of tubercle bacilli into the lung; (*3*) to relieve the patient of annoying wheezing; and (*4*) to diminish the circulation of blood in the lung and thereby prevent the general pollution of the general circulation by venous blood which has been imperfectly aerated in a poorly functioning lung. Of the 8 cases, 3 have had symptomatic relief following establishment of pneumothorax. These 8 cases suggest that pulmonary tuberculosis may be a sequel to tuberculosis of the major bronchi.—*Tuberculosis of the Major Bronchi with Little or No Manifest Pulmonary Tuberculosis, G. G. Ornstein & I. G. Epstein, Quart. Bull. Sea View Hosp., January, 1938, iii, 109.*—(*A. A. E.*)

Bronchoscopy in Tracheobronchial Tuberculosis.—Bronchoscopy has become a valuable procedure in the diagnosis of tuberculosis and the care of tuberculous patients. The lesions which are found in the tracheobronchial tree are proliferative ones (hyperplasia, tuberculoma, caseous infiltration and fibrosis) and destructive ones (ulcers and necrosis of the cartilages). Hyperplastic lesions in the bronchi may or may not be associated with caseous necrosis. Such lesions may heal by fibrosis with resultant scarring and possible stricture; or granulation tissue may be engrafted upon recent fibrosis. Ulcers begin as swollen nodules with central caseation and resultant disruption of the mucosa; they are most frequently seen in the main bronchi just beyond the carina but may appear in the trachea or elsewhere in the bronchi. Ulcers usually heal spontaneously. If they do not heal, treatment is of no avail and may even be definitely harmful. However, if the ulcer is indolent and there is no evidence of acute disease, it may be treated with mild applications of 5 per cent silver nitrate. Tuberculomata are found in the lower trachea and main bronchi; they have a tendency to undergo caseous necrosis. Treatment is necessary only when a tuberculoma is causing obstruction of a bronchus; actual cautery or coagulating electric current may be used a few times only. Fibrotic lesions constitute the intermediate and end results of all lesions of the trachea and bronchi; such lesions may cause obstruction by fibrous stricture. Dilatation is of little value because of the nature of the cicatricial tissue and the length of the stricture. In short bronchoscopy is of value chiefly as a diagnostic procedure in tuberculosis of the trachea and bronchi.—*The Limitations of Bronchoscopy in the Treatment of Tracheo-Bronchial Tuberculosis, M. C. Myerson, Quart. Bull. Sea View Hosp., April, 1938, iii, 227.*—(*A. A. E.*)

Tuberculous Empyema.—Of the 154 cases in this series, 134 had their inception between 1929 and 1933. Of 30 others included, 6 antedated this period and 3 began during 1936. Cases included were any turbid fluids in the presence of pulmonary tuberculosis or any fluid containing tubercle bacilli on direct smear. There were 47, or 30 per cent, known persistent bronchopleural fistulas and 42 known secondary infections. There were 17 cases in which empyema was not a complication of pneumothorax, 12 starting as pleurisy and effusion, and 5 by spontaneous pneumothorax. Only 8 cases occurred within a month after pneumonolysis. After fluid appeared, 67 per cent became purulent within six months and 86 became so within the first year. Six of 138 cases appeared after three years. Of 16 patients known to have had uncontaminated empyema, half became secondarily infected within the first year. Of 6 cases who had no treatment, 2 are dead, 2 are resting and 2 are well and working. Forty-six cases were treated by apiration only. Of 38 with tuberculous empyema, 15 are dead, 8 resting and 15 well and working. Of 6 cases with mixed infection, 5 are dead and 1 is resting. Of 2 cases, uncertain as to classification, one is dead and one is well and working. Seventy-six cases received treatment with dyes or saline and of 43 tuberculous empyemata, 15 are dead, 14 resting and 14 are well and working. Of 33 cases with mixed infection, 19 are dead, 9 are resting and 5 are well and working. Treatment with oleothorax was given in 35 cases and of 22 tuberculous, 6 are dead, 5 resting and 11 are well and working. Of 13 with mixed infection, 6 are dead, 4 resting and 3 are well and working. In 10 cases, 9 of which had bronchopleural fistula, a tube was inserted into the chest. Of these, 7 are dead, one is well and 2 are living. Of 22 cases with mixed infection treated in this manner, 13 are dead, 3 are well with thoracoplasty, and 6 are well with 4 who are sick and 2 improved with thoracoplasty. Sixteen tuberculous cases received thoracoplasty. Of these, 4 are dead, 7 well and working and 5 living. Of 12 cases with mixed infection who received thoracoplasty, 3 are dead, 6 well and 3 resting. There were 9 cases in which the lung was forcibly reëxpanded. Of these, 2 are dead, 3 have had thoracoplasty, 3 are living with reëxpanded lungs and one is living with the lung partially reëxpanded. Of the entire series, 76 were known to have

never had more than minimal pulmonary disease. The mortality in this group was 26 per cent. From the group of 105 cases with only tuberculous empyema, 42 are well and working whereas of the 42 who had mixed infection only, 7 are well and working. Half of those who are well have had thoracoplasty. In those cases with tuberculous empyema only, with aspiration alone, 40 per cent were well without further treatment; with the use of dyes, 9 of 14 who are well required further treatment; with oleothorax, half the cases became well without further treatment; thoracotomy is contraindicated; thoracoplasty is of value. In cases with mixed infection: aspiration alone is of no value; irrigation with salt and dyes frequently valuable in preparation for thoracoplasty; oleothorax is of no value; tube and thoracotomy frequently necessary; thoracoplasty of great value. In cases of empyema, if the lung is worth reëxpanding, try to continue with a dry pleura through frequent aspirations or the use of oleothorax. If the empyema cannot be controlled, try early thoracoplasty.—*Tuberculous Empyema, W. Woodruff, J. Thoracic Surg., April, 1938, vii, 420.*—(*L. F. B.*)

Tuberculous Peritonitis in Children.—This paper represents the accumulated cases of sixteen years in a State Sanatorium for tuberculous children and it is probable that nearly all children in Connecticut diagnosed as having tuberculous peritonitis find their way to this institution at Meriden, provided they survive the waiting period varying from a week to three months. From May, 1920 to the beginning of 1937 there were 3,125 admissions, of which 37 were diagnosed as having or convalescing from tuberculous peritonitis and 4 others under treatment for other tuberculous lesions developed it, 41 cases in all. These last afford a good insight into the onset of symptoms and clinical course. Of these 41 cases, 3 were eliminated from the study because of having left against advice within ten days of admission, leaving 38, of whom 15 were males and 23 females. Sixteen were white and 22 colored. Their ages varied from 2 to 17 years, with 25 under ten years. It is assumed that the peritoneal involvement is always secondary and Borschke is cited as having found a primary source of infection elsewhere in all but 2 of 226 cases studied. The three possible routes of invasion are (*1*) haematogenous, (*2*) lymphogenous, (*3*) by contiguity and (*4*) by rupture of tuberculous foci in abdominal organs. The first group is commonly associated with miliary tuberculosis; the lymphogenous route would include cases in which the infection follows the lymph channels to the mesenteric nodes, spreading to the peritoneum by contiguity or rupture, also the cases in which infection has extended to the lymphatics of the diaphragm from the pleura or pericardium. Infection by contiguity may have been from the intestines, adrenals or kidneys, in which case the peritonitis is usually localized. The group consequent on rupture has usually had intestinal ulceration. Calcified mesenteric lymph nodes are of special interest in view of their fairly frequent demonstration by X-ray. They are usually considered almost pathognomonic of healed or healing tuberculosis and also are a part of the picture of a tuberculous enteritis. However, peritonitis not infrequently complicates the picture and, conversely, patients known to have had peritonitis usually develop calcified nodes months or years later. Where a tuberculous peritonitis is a part of a generalized miliary infection the peritoneum will be found studded with miliary tubercles and the course is rapid and fatal within a few days. The commonest symptom presented was distension, in 22 cases. Next common were abdominal pain, 16 cases; fever, 10 cases; loss of weight, 9 cases; tumor, 8 cases; weakness, in 5; diarrhoea in only 3 and anorexia, vomiting and constipation in the remainder. Fifteen patients, or nearly 40 per cent, had had abdominal surgery, usually either diagnostic laparotomy, or laparotomy because of error in diagnosis. The remainder were diagnosed clinically. Tumor is the most interesting and difficult symptom to explain. It was usually nodular and probably due to hypertrophied lymph nodes and matted omentum. In two cases they were smooth, rounded and firm, and one of these had a faecal sinus in an operative wound, pressure causing extrusion of faeces. Six of the 38 patients presented serious constitutional complications and all of these died. There were also 9 deaths among the uncomplicated cases, a total mortality of 40 per cent. One case only had a terminal meningitis. In general the best treatment is supportive and palliative. Fluid, if present, should be aspirated according to symptomatic indications. Constipation or diarrhoea are treated symptomatically. There has recently been some enthusiasm for calcium therapy which is probably worth a trial in cases with serous exudate. Exposure by laparotomy is not recommended because the tubercle bacillus is an obligatory aerobe and the relief sometimes obtained is probably due to mechanical factors, symptoms usually returning as the gas introduced is absorbed. Prophylaxis has to do chiefly with cautioning patients with a positive sputum against swallowing

sputum.—*Tuberculous Peritonitis in Children, L. D. Van Antwerp, New England J. M., December 16, 1937, ccxvii, 995.—(A. P.)*

Tuberculous Pericarditis and Pick's Disease.—Three case reports of tuberculous pericarditis are presented. All three cases yielded tubercle bacilli from more than one part of the body, various serous surfaces being affected. One died a typical cardiac death, two died of tuberculosis. The first two cases had proved tuberculous pericarditis, the third had an enlarged heart. It is suggested that tuberculous pericarditis and the subsequent circulatory failure may be instrumental in bringing about the ascites and hepatic enlargement characteristically found in Pick's disease.—*Tuberculous Pericarditis and Pick's Disease. Report of Three Cases, Gertrude F. Mitchell, Arch. Pediat., March, 1938, lv, 157.—(M. B.)*

Jejunum in Tuberculosis.—In the course of roentgenological examination of the gastrointestinal tract of patients with pulmonary tuberculosis, certain abnormalities of the jejunal pattern were noticed. The incidence of these abnormalities has been far higher than the incidence of tuberculosis of the jejunum found at postmortem examination. The changes found have been similar to those found in patients with sprue. In this study roentgenological examination of the jejunum has been carried out in 75 patients with pulmonary tuberculosis; and in 49 of these the jejunal pattern was definitely abnormal. The high percentage of abnormalities (65 per cent) rules out the possibility of the changes being due to tuberculous involvement of the jejunum or peritoneum since at autopsy this involvement is found in only about 14 per cent of cases. It is suggested that the abnormalities in the jejunum are due to vitamin and mineral deficiencies since similar abnormalities are to be found in sprue which is due to insufficient intake of vitamins and minerals. —*Roentgen Studies of the Jejunum in Pulmonary Tuberculosis, S. Fineman, Quart. Bull. Sea View Hosp., July, 1938, iii, 433.—(A. A. E.)*

Intestinal Tuberculosis.—In autopsies performed at Sea View Hospital 70 per cent of cases of advanced pulmonary tuberculosis had intestinal involvement. However, intestinal tuberculosis occurring as the sole manifestation of organ tuberculosis is rare. This condition occurred only five times in 1,248 autopsies performed on individuals with tuberculosis. In each of these 5 cases there was extensive intestinal ulceration with no pulmonary cavitation. Two were children; 3 were adults. The intestinal ulceration may represent either a primary infection or a secondary reinfection.—*Organ Tuberculosis of the Intestine, Report of Five Cases, H. R. Nayer, Quart. Bull. Sea View Hosp., January, 1938, iii, 153.—(A. A. E.)*

Intestinal Tuberculosis.—Routine gastrointestinal examinations in tuberculosis sanatoria show an incidence of intestinal ulceration between 17 per cent (Veteran's Hospital, Sunmount, New York) to 38 per cent (Metropolitan Hospital, New York City). Brown and Sampson at Saranac Lake have made a clinical and X-ray diagnosis of ulcerative enteritis (including colitis) in 26.4 per cent of 5,542 patients with pulmonary disease. A smaller series checked by operation or necropsy verified their diagnosis in over 90 per cent. A still higher incidence of intestinal ulceration is found at necropsy in cases of fatal pulmonary tuberculosis (60 to 90 per cent, regardless of previous symptoms). In Massachusetts accurate figures are lacking because of inadequate funds for routine gastrointestinal studies of pulmonary cases, hence the reported figures are certainly too low—6.1 per cent clinical diagnoses of intestinal tuberculosis among 6,401 cases of pulmonary tuberculosis treated in institutions throughout the state. In Connecticut not a single diagnosis of intestinal tuberculosis was recorded among 2,007 cases of pulmonary tuberculosis treated from 1934 to 1936. At the Massachusetts General Hospital, since 1900, there have been diagnosed 195 cases of intestinal tuberculosis, 52 of rectal tuberculosis and 24 of anal tuberculosis. At Lakeville State Sanatorium, devoted exclusively to extrapulmonary tuberculosis, 39 cases of intestinal tuberculosis have been treated, 82 of tuberculous mesenteric lymph nodes and 1,933 of tuberculous peritonitis. State and Federal statistics are complicated by the grouping together of tuberculous peritonitis, intestinal and mesenteric lymph node tuberculosis. From 1912 to 1935 there were 2,501 deaths in Massachusetts in this group. The yearly average of 2,200 in 1930 to 1932 dropped to 1,431 in 1935. These figures obviously omit cases complicating pulmonary tuberculosis. The hyperplastic type of disease, much less frequent than the ulcerative, seldom complicates a pulmonary lesion and has been termed "primary," which is open to question, since 50 per cent of children have bovine tuberculous foci and 90 per cent of adults evidence of some tuberculous infection. The route of transmission of infection is probably both alimentary and haematogenous, with the initial

lesion in from 80 to 90 per cent of cases in the terminal ileum and caecum. The ulcerative type usually extends upward, forming circular or girdle ulcers, their edges ragged and floor covered with tubercles. The peritoneum is commonly congested and shows exudate or adhesions. Perforations are rare. Ulcers may show healing, with varying degrees of obstruction due to angulations and stenosis. The hyperplastic type of lesion predominates in the ileocaecal region, giving rise to tumor, with more or less obstruction and enlargement of adjacent lymph nodes. The onset is commonly insidious. At this stage gastrointestinal studies may be inconclusive. Later, there is nausea and vomiting and right lower quadrant pain aggravated by eating, diarrhoea, distension and marked loss of weight. X-ray diagnosis is based on Stierlin's phenomenon. Conditions to be differentiated are cancer, regional ileitis, ulcerative colitis and nonspecific granuloma. The results in the past fifteen years with the use of heliotherapy and ultraviolet radiation are compared by Moore to those with surgery in pulmonary tuberculosis, especially in the ulcerative cases. Brown and Sampson advocate it even in the presence of advanced pulmonary disease. Pneumoperitoneum has been advocated. The field of surgery is thus far limited to relief of intestinal obstruction, removal of the rare tuberculous appendix, or, in selected cases, resection of hyperplastic disease and treatment of perforation, abscesses and fistulas. The hyperplastic type of disease is amenable to either primary resection or exclusion, or exclusion may be later followed by resection with lower mortality.—*Intestinal Tuberculosis, G. A. Moore, New England J. Med., March 10, 1938, ccxviii, 418.*—(*A. P.*)

Tuberculosis Splenomegaly.—Tuberculosis splenomegaly, that condition in which there is an enlarged tuberculous spleen without any or only slight evidence of tuberculosis elsewhere in the body, is comparatively rare; only about 80 cases have been reported. Four new cases are reviewed in detail, chiefly because the changes in the blood picture throw light on the influence of the spleen on the activity of the bone marrow. The chief complaints in these cases were marked weakness and lassitude which had been present from a few months to several years. Three had jaundice of varying degree and all had slight febrile periods. Other symptoms included haematemesis, ascites, purpura, achylia, enlargement of the spleen, cough, dyspnoea and nocturnal sweating. Blood studies revealed anaemia, leukopenia, moderate thrombocytopenia with purpura coexistent in two cases. The sedimentation rate was increased in all. This is a finding not previously reported. The blood changes were so profound that diagnoses of Banti's disease, pernicious anaemia and acholuric jaundice were made. The true diagnosis was not recognized until the operation or postmortem examination was performed. Winternitz, reporting 51 cases, gives the following data regarding tuberculous splenomegaly which are essentially the same as given by other writers. It occurs in all ages, but is more frequent between twenty and forty, with the sexes affected equally. The chief symptoms are weakness, loss of weight, pains or sensation of heaviness in the left side of the abdomen. In some an enlarged spleen can be felt. Most have a moderate degree of fever and the changes noted in the blood are those found in the four cases reported. If splenectomy is not performed, the disease is fatal in from six months to fourteen years. Of 17 patients operated, 10 recovered. Other writers reported 16 cases operated, 14 of whom survived, but 7 of these later died of pulmonary or generalized tuberculosis. A few have been treated with roentgen ray with varying results. The prognosis is always dubious, but is improved by splenectomy. The diagnosis is seldom made before operation as it is readily confused with other types of chronic splenomegaly. Grossly, splenomegaly is the outstanding feature, the weight of the spleens varying between 150 to 4,000 g. The splenic involvement may be one of four types: indurated, miliary, haemorrhagic or nodular. All the cases here reported belong to the miliary form, which is characterized by a smooth surface, normal capsule, reddish-blue or purple cut surface, firm friable pulp with the pattern obscured. The miliary tubercles cannot be seen in some cases on macroscopical examination; in others where the pattern appears almost normal, close examination reveals the small nodules to be tubercles and not Malpighian bodies. Usually microscopical examination is needed to make the diagnosis. At this time a very cellular pulp with numerous, partly confluent tubercles showing little or no necrosis is seen. The Malpighian bodies are few. The nodules in cases reported were present along the arteries, mostly being intimately connected with the Malpighian bodies. Numerous red cells were present, but no increase in phagocytosis or blood pigment was noted and the connective tissue was not increased. Much of the splenomegaly is due to proliferation of the reticulum cells in the pulp and the presence of tuberculous foci. The blood changes are of especial interest. In miliary tuberculosis

with splenic involvement the changes are those usually observed in exosplenic tuberculosis. However, in tuberculous splenomegaly there are significant changes in the blood picture closely resembling that of Banti's disease. Occasionally polycythaemia is found, but most cases are reported as showing anaemia, leukopenia and thrombocytopenia. In the cases reported by the author many blood studies were made, including bone marrow examination in two cases; in one case, the blood changes associated with acholuric jaundice were present. This probably was a complicating factor. The other cases showed a low red blood count, low haemoglobin, color indexes of .82 to 1.2, low platelet count and an icterus index of 5 and 10. Thus all patients showed one or more signs of inhibition of the bone marrow function. The rôle of the spleen is conspicuously seen in case 2 in which the patient, though apparently hopeless, survived a splenectomy following which the haemoglobin, red blood count, white blood count and platelets became normal or nearly so. The sedimentation rate remained rapid, probably because tuberculosis of the spleen was not the only focus. Study of the bone marrow three months after the operation revealed a marked decrease in immature cells and also the number of mitoses, whereas before operation the bone marrow showed a relatively immature picture with shift to the left. The relation of the spleen to the bone marrow function is in question. Some believe that the normal spleen does not have an inhibitory effect on the bone marrow, while others have concluded that it does. Most observers have noted an increase in the white blood count and platelets following splenectomy, and frequently immature erythrocytes and leucocytes are seen. Frank pointed out the fact that leukopenia and thrombopenia follow an increase of reticuloendothelial elements in the spleen; the author's cases support this view. The changes in the blood picture and bone marrow before and after splenectomy suggest that there is a controlling function by the spleen on the emission of cells from the bone marrow. In support of this, attention is called to similar changes produced in "fibrosplenia" of Banti's disease and certain other splenomegalies such as malaria, reticulum cell sarcoma and Gaucher's disease. The author thinks it not unreasonable to assume that the spleen has a controlling function on the emission of cells from the bone marrow, this function probably originating the reticuloendothelial elements, especially the reticulum cells. He concludes that "splenomegalies caused by miliary tuberculosis, like certain other splenomegalies where proliferation of the reticulum cells has taken place, may give rise to an apparent inhibition of the bone marrow function, with subsequent peripheral blood changes" and "that these blood changes may probably be interpreted as the result of an inhibition of the emission or of the maturation of the blood cells in the bone marrow, but not of the production of the cells there."—*A Study of Tuberculous Splenomegaly and Splenogenic Controlling of the Cell Emission from the Bone Marrow, J. Englebreth-Holm, Am. J. M. Sc., January, 1938, cxcv, 32.*—(*G. F. M.*)

Tuberculous Disease of Abdominal Lymph Nodes.—A study of 239 consecutive cases of caseous and calcareous abdominal lymph nodes showed that the average age of those with calcareous nodes was twenty-five years. Calcareous nodes were much more common than the caseous ones. Most of the cases were due to the bovine type of the bacillus. At operation no definite bowel lesion was seen in any case. Twenty-five gave personal or family histories of tuberculosis, but in only 3 was tuberculosis found in other locations than the abdomen. The most common symptom of this condition was pain. The pains came on suddenly and often disappeared just as suddenly. There seems to be considerable disagreement as to the explanation of this pain. Ten cases had an acute intestinal obstruction and 3 of these died. When intestinal obstruction occurred the symptoms were typical of that condition. There were also 3 cases that had a subacute obstruction. The appendix was found to be tuberculous once in the 93 cases in which it was removed. In operating on these cases a right paramedian incision was usually used. Treatment consisted of removal of the caseous or calcareous nodes. After the removal of the nodes the mesentery should be carefully sutured. The postoperative results were as follows: excellent in 148 cases, good in 24, poor in 14, bad in 15, and 10 died. Of these deaths only 5 can be classed as operative mortality.—*Some Surgical Aspects of Tuberculous Disease of the Abdominal Lymphatic Glands, G. H. Colt & G. M. Clark, Surg., Gynec. & Obst., December, 1937, lxv, 771.*—(*H. W. H.*)

Prognosis in Tuberculosis of Tonsils and Adenoids.—An attempt was made to locate 79 individuals who had had tuberculous tonsils or adenoids removed between 1912 and 1920. Out of these 79 cases, information was obtained concerning 61. Of these, 7 had died, 37 returned for hospital

examination, and 17 gave information either by letter or by interview with a social worker. The time interval between operation and final note with three exceptions ranged from fifteen to twenty-three years. Significant data are presented in tabular form. The patients are classified in two groups, one including those under fourteen years of age at the time of operation and the other all those older than fourteen at operation. It should be borne in mind that these patients did not have clinically recognized tuberculosis of the tonsils and adenoids. The lesions were found in the excised material and were believed to be incidental to a more widespread infection. In 44 of the 61 tabulated cases the cervical lymph nodes were considerably enlarged at the time of the original operation, and in 23 cases a diagnosis of tuberculous adenitis was made. Unfortunately, the preoperative studies were too incomplete, and roentgen ray examinations were too sporadic to allow one to estimate, in retrospect, the incidence of pulmonary tuberculosis at the time of discovery of the tonsillar lesion. The available evidence indicates that a considerable number of the older patients had pulmonary tuberculosis at the time of the operation, and a fair proportion of the younger patients showed evidences of a childhood type of tuberculosis. In a much larger proportion of this group there was clear evidence that the cervical lymph nodes were involved. Of the 47 patients operated upon prior to the age of fourteen, 45 could be traced. Two had died of nontuberculous diseases, and one of tuberculous meningitis several months after operation. All of the 42 living patients were well and there was no evidence that any of them had developed tuberculosis of the adult type. Of the 32 patients operated when past the age of thirteen years, only 16 could be traced. One had died of nontuberculous disease. At least 2, and possibly 3 had died of pulmonary tuberculosis. Of the 12 patients known to be alive, one has progressive pulmonary tuberculosis and 3 others have fibroid apical disease. Two additional patients at one time had physical signs of apical tuberculosis. Five patients gave a history of pleurisy. Of the 16 patients who could not be traced, 3 had intrathoracic tuberculosis at the time of operation. The available evidence indicates that the relatively poor prognosis in the older group was attributed to the existence, at the time of operation, of pulmonary tuberculosis of the adult type. When tubercles are discovered in the tonsils, adenoids or cervical lymph nodes of preadolescent children the immediate prognosis apparently depends upon the character of the disease elsewhere in the body. Death may occur from the progress of the existing childhood disease, but if the patient recovers from the latter, he is apparently not likely to develop adult forms of tuberculosis during the ensuing fifteen to twenty years. The complete absence of progressive pulmonary tuberculosis among the preadolescent patients with tuberculous cervical adenitis is in keeping with Marfan's observation that individuals whose scrofulous lesions heal prior to the age of fifteen rarely develop progressive tuberculosis in later life.—*Prognosis in Cases of Tuberculosis of the Tonsil, Adenoids and Cervical Lymph Nodes. A Study of Patients followed from 11 to 23 Years, J. Bordley, III & J. W. Baylor, Bull. Johns Hopkins Hosp., September, 1938, lxiii, 132.*—(*J. S. W.*)

Tuberculous Cervical Adenitis.—Tuberculosis of the lymph nodes tends to run a long and debilitating course but with a disposition to undergo natural cure when the resistance can be raised by circumstances and environment. Reid and Wilkinson set forth a method of treatment based on underlying pathology that has achieved fair success within reasonable time. They review 119 consecutive cases of tuberculous cervical adenitis treated in Black Notley Sanatorium (Essex County Council) over a period of six years. The cervical nodes drain the tonsil and lymphatic tissue of the nasopharynx. Howarth and Gloyne, some fifteen years ago, found the tubercle bacillus present in the tonsil in about 5 per cent of 300 cases whose tonsils were removed and who also showed cervical adenitis. Of the present series of 119, 85 patients had a history or signs of tonsillar disease, in 21 only was the throat normal, and 31 had tonsillectomy prior to admission, while 22 had tonsils and adenoids removed during their stay. The upper carotid chain is most often involved but nodes of the posterior triangle and supraclavicular region are not uncommonly so, and the disease often is present in more than one cervical group. In no case has the disease been found to reach beyond the lymphatic system nor has any case shown secondary pulmonary or bone tuberculosis. If there is caseation rupturing the gland capsule it may burrow, penetrate the deep fascia and become subcutaneous, a "collar stud" abscess which if let alone will burst. In favorable cases the stage of fleshy enlargement retrogresses and the node shrinks to normal. In a rarer type many nodes are involved in a fashion resembling Hodgkin's disease and these are multiple, discrete, mobile and do not caseate. They tend to recur after removal and are not responsive to

constitutional treatment. At any stage, as natural resistance becomes effective, resolution may begin. It is the more persistent and difficult cases that require radical operation, for example, those residual infections following natural or artificial attempts at cure. Tuberculous lymphadenitis is to be regarded as a local tuberculous lesion in a patient with insufficient resistance. Constitutional treatment is paramount and the chief factors are rest, open air, a liberal balanced diet with adequate vitamins, and heliotherapy. Physical activities should be limited, but total bed-rest is not necessary unless the disease is very active. Both natural sunlight and artificial phototherapy are of value and Wilkinson has seen cases of tuberculous lymphadenitis treated successfully by radium in conjunction with constitutional measures. Where there is evidence of tonsillar disease the tonsils are removed as soon as the general condition warrants it. Tuberculous abscesses are managed according to whether there is superadded secondary infection or not; if so, incision and free drainage are necessary; if not, aspiration should be tried; however, the success of aspiration in tuberculous abscesses generally is dependent on access through and across muscle, contraction of muscle fibres closing the needle track (Gauvain). In the neck anatomical conditions are not favorable for this and there are difficulties in removing the pus, which often accumulates. It is usually simpler to empty the abscess through a small incision with gentle curettage and suture of the wound. The risk of secondary infection is small, periadenitis often quickly subsides and later radical resection is facilitated. Radical excision enters a controversial field and there have been many poor results, such as recurrence, persistent sinus, muscle pain and ugly scars. These can usually be explained by too early operation or lack of general care and treatment. Periadenitis is a contraindication, also very active progression of the process. Transverse incisions are always to be preferred. If there is a visible sinus a director is passed into it and the node pulled up. The sternomastoid muscle is dissected backward and the fascia and vessels then peeled off. High up the spinal accessory nerve is encountered and must be carefully preserved. Dissection and traction are always upwards and all except soft seedling nodes removed. Drainage posteriorly for twenty-four hours is used. Immediate results were good in all groups except 5 cases, which should have been completed by operation. The late results also were generally satisfactory, no fresh foci having developed in any case. Of 8 cases not so, definite reasons were discovered in 6, insufficient constitutional treatment in 3, insufficient surgery in 2, failure to carry out tonsillectomy in one.—*Tuberculosis of the Cervical Lymphatic Glands, B. Reid & M. C. Wilkinson, Brit. M. J., October 16, 1937, p. 740.*—(*A. P.*)

Tuberculous Cervicitis.—Eighteen cases of tuberculous cervicitis are reported, in one of which this was the only active tuberculous focus present. Very few cases have been reported in the past, but a survey of the literature shows that 90 per cent were secondary to upper genital tract tuberculosis, particularly the Fallopian tubes, the remainder of the cases having been secondary to tuberculous foci in the lungs, intestines or skeletal system. In the absence of a demonstrable tuberculous focus, tuberculous cervicitis may indicate genital tuberculosis in the marital partner. The disease occurs generally in the twenty to forty year age group with predilection for the American negress. Persistent offensive watery leucorrhoea and postcoital bleeding are the most common symptoms. The cervix most frequently assumes a papillary or ulcerative form, but endocervical or interstitial forms are occasionally seen. It is less friable and more injected than carcinoma of the cervix and is symmetrically hypertrophied. Spread to the vaginal fornices, endometrium and broad ligaments is by direct extension and slow. On biopsy, the presence of bacilli, tubercles and giant cells in the mucosa and stroma confirm the diagnosis. The treatment of choice is panhysterectomy with removal of both fallopian tubes. Ultraviolet radiation and sanatorium care are recommended for inoperable cases. Because it resembles cervical carcinoma and because it may reveal genital or extragenital tuberculosis, the clinical recognition of tuberculous cervicitis is important.—*Tuberculosis of the Cervix, C. S. Stevenson, Am. J. Obst. & Gynec., December, 1938, xxxvi, 1017.*—(*A. B. C.*)

Scrofula and Immunity in Tuberculosis.—In tuberculosis some viable bacilli must be constantly present in the organism to confer immunity. "Premunition" is defined by the author as resistance to reinfection in persons having any chronic disease. Scrofulous lesions are relatively benign in character and confer a certain degree of resistance to tuberculosis. Both allergy and immunity may appear and disappear simultaneously. Allergy probably plays a minor rôle in the defense mechanism. It is only one of the many factors which make for immunity in tuberculosis. According to Ranke's theory, the older the

lesion, the higher the allergy and the greater tendency to localization. Scrofulous lesions in various organs are metastasis by way of the blood-stream from an initial focus. The lesions are not necessarily produced by the bovine strain of tubercle bacilli or by the so-called tuberculous ultravirus. When numerous bacilli enter the circulation fatal dissemination occurs. With fewer bacilli and high immunity, healing takes place; at times a chronic scrofulous lesion may result. The form and evolution of scrofulous tuberculous lesions vary with number of bacilli present and relative state of immunity at time of invasion. Vascular stasis may often explain the localization of bacilli for certain organs.—*La scrofule, forme spéciale de la tuberculose avec des remarques sur l' immunité antituberculeuse, A. B. Marfan, Ann. Inst. Pasteur, October, 1938, lxi, 325.*—(*J. G.*)

Phlyctenulosis.—The yearly incidence of phlyctenulosis in 1935 and 1936 was, according to hospital statistics in Boston, but 7.5 per cent of that twenty-five years previously. The decrease is greater than the decrease in the annual incidence of pulmonary tuberculosis; it more nearly approximates that in the incidence of nonpulmonary tuberculosis. Of 502 children for whom a clinical diagnosis of phlyctenulosis was made, 300 gave positive reactions to the tuberculin test. One hundred and nine showed clinical or roentgenological evidence of tuberculosis. In all of these the tuberculosis was of the primary or "childhood" type, and in all but 3 cases the condition was relatively mild. Girls seem more likely to have phlyctenulosis than boys. The nutritional condition of the children with phlyctenulosis as a group was normal. There is considerable evidence in our data that a phlyctenule is an allergic manifestation in a patient whose reaction to tuberculin is positive; for the reaction when positive is strongly positive, phlyctenules have not been observed in anergic tuberculous patients, and phlyctenules are rarely seen in the very sick tuberculous patient. There is no evidence in our data that a phlyctenule is an allergic manifestation in a patient whose reaction to tuberculin is negative; in fact, such patients are usually not extremely atopic. Phlyctenulosis should probably be divided into two types, the tuberculous and the nontuberculous. Clinical diagnosis, without a tuberculin test, is insufficient at present for differentiation between these two types. The cause of the nontuberculous type of phlyctenulosis has not been satisfactorily explained.—*Phlyctenulosis, L. B. Burgin & H. L. Higgins, Am. J. Dis. Child., August, 1938, lvi, 239. (Authors' Summary)*—(*M. B.*)

Cerebral Tuberculosis Simulating Tumor.—A boy of seventeen years who at the age of six years had had convulsive spells showed, after ten years of freedom from symptoms, evidence of hypertension, epileptic seizures, papillary oedema and loss of the patellar and left Achilles reflex. Slight rigidity of the spine and irregular subfebrile temperatures were observed. The symptoms were interpreted as due to a cerebral tumor of the posterior cerebrum; a tubercle of the occipital region was thought of. The patient died suddenly and the autopsy revealed conglomerate tubercles in both the anterior third of the left hemisphere and in the posterior lobe. The author assumes that the tuberculosis had developed in a rather slow course probably beginning ten years before death and that the sudden death must be attributed to extensive tuberculous softening and oedema of almost the entire white matter of the left hemisphere. There was no evidence of meningitis and, therefore, the slight stiffness of the neck probably was directly caused by the tubercles of the posterior lobe. Histological findings revealed an abundance of vascular thrombosis and of sclerosed connective tissue, of tuberculous granulations and of softening.—*Tuberculose cérébrale simulant une tumeur, C. Urechia, Bull. de l'Académie de méd. de Roumanie, May, 1938, v, 328.*—(*F. G. K.*)

Calcified Tuberculoma of Brain.—Three cases are added to the 15 found in the literature. The occurrence of calcification or ossification in tubercles or tuberculomata of the brain is relatively rare and is indicative of partial or complete healing of the lesion. In a series of 15,000 autopsy records there were 405 cases listed as tuberculous meningitis and 43 cases of tuberculoma of the brain. In 3 cases, about 7 per cent, the tuberculomata were either calcified or calcified and ossified. Calcification or ossification may also occur in miliary meningial tubercles, representing a healed tuberculous meningitis, and was present in one of the cases reported. It does not necessarily occur in all tubercles or tuberculomata in a given case. Although death usually occurs before calcification can take place, the quiescence of such tubercles is evidenced by some patients' long period of survival, in one case over sixty years, and in a number for one and a half to eleven years after the condition was diagnosed. The process of calcification is preceded or accompanied by more or less complete hyalinization of the peripheral zone of granulation tissue. The calcium is deposited in the periphery of the central necrotic area and beneath the connective

tissue capsule, forming a calcareous shell. Whether this occurs in dead tissue, before or after fibrosis and hyalinization, is not known. The shell is irregular in contour and often broken, probably as a result of contracture of the capsule. It is believed that the irregular, broken calcareous shell is characteristic of the deposit of calcium in tubercles. The mechanism of calcium deposition is uncertain but may be the result of impaired tissue respiration, due to the presence of fatty acids in the decadent tissue, or may be formed by hyalinization of the adjacent connective tissue. Accessibility to the circulation appears to be necessary. Bone formation is less common than calcification and was present in one case. It suggests that the presence of calcium favors the formation of bone. The bone has the characteristics of new-formed osteoid tissue in which calcium has not yet been deposited.—*Calcification and Ossification in Tuberculoma of the Brain, H. S. Evans & C. B. Courville, Arch. Surg., April, 1938, xxxvi, 637.*—(*L. F. B.*)

Variability of Findings in Tuberculous Meningitis.—A total of 135 cases of tuberculous meningitis were admitted to the Children's Hospital in San Francisco from 1920 to 1937. There has been a general decline in the numbers of such admissions during the past six or seven years. More cases are always admitted during the winter season. The greatest number of cases were less than three years of age. Vomiting and fever were the most frequent complaints, and convulsions and drowsiness were less frequently complained of. On physical examination all 135 had fever. Ninety-four had a definitely stiff neck, and 54 had a positive Kernig. Forty-seven were admitted unconscious, and convulsions occurred in 30. One hundred four of the 135 had chests negative to ordinary physical examination. X-ray films of the chest were taken in only 85 of the cases. Eleven X-ray films were read as negative; miliary tuberculosis was found in 21. Tuberculin was applied in 70 of the cases and there were 38 positive reactions; the remaining 32 were negative to 1:1000, no second tests being made. Blood counts contributed nothing to the picture. The most useful diagnostic aid was examination of the spinal fluid. Such examinations were made on 128 of the cases; 102 showed cell counts ranging from 50 to 200 cells; 16 cases had less than 50 cells, and 10 had 500 or more. In 60 per cent of the cases studied, more than 75 per cent of the cells seen in the spinal fluid were lymphocytes; in the remainder they constituted 50 per cent or less of the cells counted. Tubercle bacilli were identified in the spinal fluid in 62 per cent of the cases; in 38 per cent they could not be found. As to duration of the disease, the greatest number of cases, 58, lived less than two weeks, while 46 lived between two and four weeks after onset; 4 survived more than four weeks. In 27 the duration of the disease was not known.—*Variability of Findings in Tuberculous Meningitis, H. E. Thelander & Janet Baldwin, Arch. Pediat., April, 1938, lv, 225.*—(*M. B.*)

Tuberculous Meningitis Resembling Diabetic Coma.—There is reported a case of a three and one-half year old Puerto Rican girl whose paternal grandfather had died of diabetes. She gave no history of contact with tuberculosis. She presented a fresh gonococcal vaginitis and a paronychia of the right index finger. Temperature was about 101°F. Mantoux test was negative with a dilution of 1:1000. The urine was normal on admission. About three days after admission, the child became extremely drowsy, respirations were irregular, but neurological examination was negative and spinal fluid appeared normal. The urine however showed four-plus sugar and three-plus acetone. The blood sugar showed a level of 200 mg. and the carbon dioxide combining power was 36. Treatment for diabetic coma was instituted and resulted in insulin shock with no relief from drowsiness. Convulsions began about four days after the onset of drowsiness and during a period when the blood sugar was normal. At that time the spinal fluid showed findings typical of tuberculous meningitis, and the Mantoux test repeated with a dilution of 1:100 was positive. The child died thirteen days after admission and tuberculous meningitis associated with tuberculosis of the tracheobronchial lymph nodes was found at autopsy. The pancreas appeared essentially normal. The confusing hyperglycaemia may be explained by the involvement of the glycogenic centre at the floor of the fourth ventricle, and the acidosis is believed to have been due to starvation and bouts of vomiting.—*Tuberculous Meningitis Resembling Diabetic Coma, L. L. Krafchik & L. B. Slobody, Arch. Pediat., May, 1938, lv, 288.*—(*M. B.*)

Treatment of Joint Tuberculosis.—The author reviews the accepted methods of general, conservative and surgical treatment for tuberculosis of bone and joints. Advances in treatment during the past twenty years are such that mortality under treatment has been greatly reduced and arrest of disease takes place in 80 to 90 per cent of patients admitted to special hospitals.—*The*

Treatment of Tuberculosis of the Joints, J. G. Johnstone, J. Roy. Inst. Pub. Health & Hyg., May, 1938, i, 453.—(L. F. B.)

Bone Tuberculosis.—The controversial theories concerning the relation of trauma to the development of bone tuberculosis are reviewed and discussed. The theories advanced to explain primary bone tuberculosis as the result of trauma on the basis of tubercle bacillaemia, *locus minoris resistentiae*, and hyperaemia are shown to be untenable. Experimentally, the author failed to produce bone tuberculosis at the site of artificially induced fractures in tuberculous rabbits. The absence of bone tuberculosis in the injured during the World War and following surgery, either in general practice on patients who, it is safe to assume, may occasionally have latent tuberculosis, or on known tuberculous patients when thoracoplasties are performed, are additional arguments. These facts and recent investigations which revealed that the majority of cases of bone tuberculosis are associated with other old, inactive bone foci, lead the author to conclude that trauma does not produce primary bone tuberculosis, but rather that a latent tuberculous bone focus can manifest itself through trauma.—*Knochentuberkulose und Trauma, H. O. F. Haberland, München. med. Wchnschr., August 19, 1938, lxxxv, 1257.—(A. B. C.)*

Skin Temperatures in Tuberculosis of Joints.—Investigations of the skin temperatures in about forty patients with joint diseases, mostly of tuberculous origin, have been conducted by the method of Ypsen and Foyed. Special mercury thermometers are placed under a small piece of felt at symmetrical points above, laterally, medially to and below the joints. In normal cases the difference between the temperatures at symmetrical points is small and ranges between zero to 0.5 and 1.0°C., seldom more. In patients with tuberculous joint lesions the differences of the skin temperature often will be from 1 to 2°C. up to 4°C. One case showed an elevation of 8.5°C. The observations included patients of all stages of tuberculosis and also of various stages of different treatments. The differences proved to be consistent with the character of the disease, being small in quiescent cases and higher in more advanced cases. After successful treatment the differences showed a marked diminution and after the disappearance of clinical symptoms the differences were very small. In patients with other than tuberculous infections the skin temperatures showed only small differences. Sometimes marked differences were observed while the clinical signs were rather vague. Later on, clinical course and X-ray findings showed an active tuberculosis in these cases, as was previously suggested by the result of the skin temperature test. Greger points to the practical value of the test in both diagnosis and differential diagnosis of tuberculous joint lesions.—*Investigations of the Skin Temperature in Patients with Diseases of the Joints, C. Greger, Acta orthop. Scandinav., June, 1938, lx, 72.—(F. G. K.)*

Tuberculous Infection of the Hip Joint.—An attempt was made to determine the point of primary involvement in 82 cases of tuberculosis of the hip that were studied roentgenologically and 56 that were studied pathologically. The earlier after the onset of symptoms the case was studied the greater the chance of discovering the primary point of infection. In 70 cases the disease began in childhood and in 12 cases in adult life. In those cases beginning in childhood the primary point of infection was determined in 26 cases; neck of the femur adjacent to head, 14; ileum bordering on acetabulum, 10; ischium bordering on acetabulum, 2. In the 44 remaining cases that began in childhood the primary point of infection could not be determined. In the 12 patients in whom the disease began in adult life, the lesion was diagnosed as primary in the greater trochanter in 2, in the ilium in one and in the other 9 it was impossible to determine the primary site.—*The Primary Point of Infection in Tuberculosis of the Hip Joint, C. H. Hatcher & D. B. Phemister, Surg., Gynec. & Obst., December, 1937, lxv, 721.—(H. W. H.)*

Tuberculous Osteitis of the Ischium.—From 1928 to 1936, 8 cases of tuberculous osteitis of the ischium, representing 0.2 per cent of all cases of bone tuberculosis, were treated. Six were males and 2 were females. The left ischium was involved in 5 cases and the right in 3. Five other cases of uncertain aetiology were treated during this time. The course of the disease is insidious and protracted, the first symptoms being usually slight pain of the sciatica type. The patients were usually able to continue working. Later localized pain over the tuber ischii was present especially when the patient was seated. Later abscesses or fistulae appeared in the gluteal region. In 3 cases there were symptoms of hip joint disease as stiffness of the joint, pain radiating down the leg and slight restriction of motion. In these cases the destruction within the ischium reached the acetabulum although in other cases the degree of bone destruction was similar but the patients were without hip joint symptoms. In all cases bone destruction was

localized to the lateral part of the superior ramus or the lower part of the tuber ischii, and did not show any tendency to ascend and break through into the hip joint. Involvement of the hip joint not infrequently descends along the superior ramus of the ischium. In diagnosis, specific inflammatory process, tumor, and syphilis must be considered. Roentgenologically septic osteitis seems to leave a more sclerotic bone delineation and sharper outlines of destruction than does tuberculous osteitis. Of the 8 cases, one was treated conservatively, 3 had only sequestrectomy, 2 were scraped out and in 2 subperiosteal resection of the tuber ischii was performed. Although fistulae were found in 5 cases at operation and some appeared after operation, all but 2 were healed when discharged from the sanatorium. These patients have remained healed, from one to eight years, and are otherwise symptom free.—*Tuberculous Osteitis of the Ischium, R. Magnusson, J. Bone & Joint Surg., January, 1938, xx, 23.*—(*L. F. B.*)

Sacro-iliac Tuberculosis.—A study is presented covering the history of 23 cases of sacro-iliac tuberculosis treated by operative fusion, which has been the method of choice since 1920 in every instance permitted by the condition of the patient. Of the 23 patients,13 were females and 10 were males. The average age at onset was twenty-three years, with 4 patients younger than fifteen. Sacro-iliac tuberculosis is one of the rare forms of tuberculosis and usually indicates a widespread infection in the body. Forty-three per cent had pulmonary tuberculosis, and 35 per cent had lesions in the vertebral bodies as well as in the sacro-iliac joint. The average duration of symptoms before operation was two and one-half years, the longest fourteen years and the shortest three weeks. All of the patients had pain. Most commonly it was a dull ache in the buttock, frequently described as "a pain in the hip." This was present to a greater extent on standing and walking than lying. Only 7 patients had sciatica. It was always on the side of the affected joint. An interesting feature of this sciatica was that 5 of the 7 patients had a large abscess in the buttock, causing tension or inflammation of the ligaments, fascia and gluteal muscles. Disease of the sacro-iliac joint in itself does not tend to produce pain of the sciatic type. The preoperative diagnosis was not easy. Clinical signs and symptoms suggested disease of the joint but were in no way pathognomonic of tuberculosis. Roentgenograms and laboratory tests also offered no conclusive evidence. All these factors, however, indicated the need of exploration of the joint for possible tuberculosis. Arthrodesis of the joint was usually accomplished by a posterior approach with the patient under ethylene anaesthesia. No attempt was made to obtain all the cartilage or carious bone from the joint, since arthrodesis and healing were accomplished in all but one case even though surgical invasion of the joint was minimal. The neighboring sides of the sacrum and the ilium were chipped up, forming small and large, free and attached bone chips. Additional bone, if needed, was taken from the external surface of the ilium. After the operation the patient was left in bed with a spine brace in some instances and with no support at all in others. A plaster cast was not used. In 12 cases draining sinuses developed after the operation. Most of them closed spontaneously within five months. Two sinuses drained for over five years but have since healed completely. The length of the follow-up period varied from six months to ten years. The average was 4.9 years. Eighteen patients are believed to be healed of their sacro-iliac tuberculosis; 17 have been actually cured and restored to active work and usefulness, several for as long as eight or ten years. One patient with tuberculosis of both hips is still under treatment, although it is three years since solid fusion of the sacro-iliac joint occurred and she is free from her sacro-iliac pain. The length of stay in bed after operation varied, depending on whether tuberculosis was active elsewhere in the body or whether the focus was quiescent. Sixteen patients in group 1 were kept in bed until, clinically, the activity had abated. This required sometimes as long as three years. The average length of stay in bed after operation for the 7 patients in group 2, who did not have an active demonstrable focus elsewhere in the body, was three months. Although this interval seems somewhat radical for patients with tuberculosis, this is not the case with tuberculosis of the sacro-iliac joint. It is not necessary to insist on rest in bed until, as shown by roentgenograms, the diseased area is homogeneous bone. Walking and even the commencement of work did not in a single case in this series cause a recession in healing of the bone at the area of the disease. That sacro-iliac fusion results in cure of the local tuberculosis is shown by the fact that, when once the joint is solid and the bone has attained a healthy appearance in roentgenograms, a recurrence has never been noted. Five patients died, all within one year after operation.—*Sacro-iliac Tuberculosis, F. Thompson, J. A. M. A., May 7, 1938, cx, 1538.*—(*G. L. L.*)

Spinal Fixation.—The author prefers a modification of the Albee method wherein the graft is laid beneath the *erector spinae* muscles in the junction of the bases of the vertebral spines and the laminae, the cancellous portion of the graft being opposed to the freshened bone. When a flexible graft is advisable or necessary it is produced by making from two to four longitudinal cuts to within two inches of the bone of the graft. When in place, the deep layers of the interspinous ligament are sutured to the deep layer of the muscles, making the stitches quite close together. As the suturing progresses, the graft bends and fits over any marked gibbosity.—*Spinal Fixation, G. Hamada, J. Bone & Joint Surg., January, 1938, xx, 85.*—(*L. F. B.*)

Spinal Fusion in Tuberculosis of Spine.—Three hundred nine children under ten years of age with active tuberculosis of the spine have been treated by spinal fusion and 74 per cent of them have had good results. Forty-five per cent completed their hospitalization and need for support in ten months.—*The Treatment of Tuberculosis of the Spine by Spinal Fusion. Results in 309 Children less than Ten Years of Age, W. E. Swift, J. Pediat., August, 1938, xiii, 248.*—(*M. B.*)

Tuberculous Spondylitis.—The author has studied the roentgenological development, localization and appearance of different forms of healing on a material of 614 spondylitic patients. Tuberculous spondylitis often extends directly to adjacent vertebrae over the intervertebral discs and destroys several vertebral bodies in succession. The tuberculous lesion progresses subperiostally along the anterior or posterior surfaces of the bodies and invades its central parts by following the vascular route, thus showing progressive infiltration rather than haematogenous spread. The following forms of healing can be established: (*1*) Paradiscal spondylitis with the tuberculous lesion chiefly located in the parts of the body adjoining the cartilage. While in a few cases the bony lesion is well defined and circumscribed and presents a semilunar or rounded appearance there is in other cases a fairly extensive area of bone destruction of the vertebral margin with more or less marked partial vertebral collapse. (*2*) The spondylitic lesion appears on the film merely as a narrowing of the intervertebral disc which is probably due to direct invasion of the cartilage from a superficial bone lesion, the latter being too small to be visualized on the film. (*3*) Spondylitis with continuous extensive destruction of smaller or larger parts of the spine involving two or more successive vertebral bodies. (*4*) Central type of spondylitis where the lesion does not reach the vertebral periphery. (*5*) In the vesicular type of spondylitis bleb or cystlike areas of decreased density are scattered throughout the vertebral body. (*6*) Anterior, posterior and lateral spondylitis. (*7*) Spondylitis with diffuse osteoplastic changes. Among the material observed there were multiple primary bone lesions of the spine in 9 per cent. Tendency of bridging is largely dependent upon the type of spondylitis and this form is more frequent in the fairly common paradiscal type than in any other. This type is fairly rare in childhood and occurs frequently in the older age groups. The subperiosteal spurs and bridges are due probably to direct stimulation from the bone lesions near the adjoining vertebral bodies and from paravertebral abscesses rather than from changes in the dynamic conditions as it is assumed in deforming spondylosis. Spastic paralysis was seen particularly often in cases of extensive destructive spondylitis and it occurred fairly frequently during the first ten years of life.—*Ueber die Entwicklung des tuberkulösen Processes und die Entstehung verschiedener Ausheilungsformen bei Tuberkulose der Wirbelkörper, A. Hellstadius, Acta orthop. Scandinav., January, 1938, viii, 1.*—(*F. G. K.*)

Hospital Observation on Spondylitic Patients.—The object of the studies was to attempt to show how 237 patients treated in the Coast Hospital Stavern in Norway for tuberculous spondylitis have fared over a period of eighteen years. The author gives a survey of the localization of the lesion in children and adults, of further spread of the disease to other organs and of the course of the death rate both at the Coast Hospital and after discharge with reference to the age of the patients and to the localization of the lesion. It has been the author's especial endeavor to divide the material into cases treated with corset and those treated with plaster jacket application and to follow the course and late results of these two groups. The results are based on replies received from the patients themselves, partly from their families and relatives. Pulmonary tuberculosis as a late complication played a most important rôle and developed in 20 patients during the hospital stay. The pulmonary lesion which in 3 cases was of the miliary type probably always represents a flare up of previous pulmonary lesions. While the spondylitic process in childhood is more often in the cervical and dorsal spine, in adults lumbar spondylitis is more frequently

seen. The death rate during the hospital stay was 19.6 per cent for children, and 9.5 per cent for adults. Following discharge from the hospital 24.4 per cent children and 22.4 per cent adults died, thus bringing the total death rate to 36.3 per cent in children and to 32.9 per cent in adults. Plaster jacket treatment as far as adults are concerned gives a strikingly successful result and seems to be more encouraging than the results following the application of the corset treatment.—*Nachuntersuchungen von Patienten behandelt wegen Spondylitis tuberculosa auf dem Seehospital bei Stavern 1911–1929, A. Grevle, Acta orthop. Scandinav., January, 1938, viii, 304.*—(*F. G. K.*)

Short Bone Graft for Spinal Fusion.—The author proposes the name spondylodesis for the use of a short bone graft for a tuberculous spine because of the analogy to arthrodesis. The fusion of only the affected vertebrae is advocated and the method is used as a final procedure of conservative treatment. It is thought that the long bone graft interferes with the natural correction of the parafocal spine. If done early, it cannot arrest the extension of the disease and interferes with the process of repair in that it impedes collapse and elimination of space between the destroyed vertebrae. More motion of the spine is allowed by the short graft but this motion is in the normal intervertebral discs above and below the lesion.—*Spondylodesis, J. De Mol Van Otterloo, J. Bone & Joint Surg., April, 1938, xx, 320.*—(*L. F. B.*)

Intra- and Extrathoracic Tuberculosis.—This paper presents an analysis of lesions found in 976 tuberculous individuals discharged from Glen Lake Sanatorium, Oak Terrace, Minnesota. The presence or absence of pulmonary involvement was determined by X-ray examination and tuberculin test, and the cases (976 proved tuberculosis) classified into 6 groups: (*1*) far advanced pulmonary tuberculosis, 54 per cent; (*2*) moderately advanced pulmonary tuberculosis, 25 per cent; (*3*) minimal pulmonary tuberculosis, 8 per cent; (*4*) childhood type tuberculosis, 6 per cent; (*5*) pleurisy with effusion, 0.5 per cent; and (*6*) pulmonary tuberculosis not demonstrable, 6.5 per cent. All but 11 cases were regarded as clinically active. Laryngeal and enteric tuberculosis are the two commonest extrapulmonary complications of pulmonary tuberculosis, and in the past more common and severe than usual to-day. They are almost always associated with far advanced or moderately advanced pulmonary disease. In this series 20 per cent and 4 per cent respectively, and a total of 13.5 per cent of all pulmonary cases were recorded as having laryngeal lesions. Tuberculous enteritis was recorded in 14 per cent of far advanced and 6 per cent of moderately advanced cases and in one of 78 minimal cases, or altogether, in 10.6 per cent of all pulmonary cases. Skeletal tuberculous lesions occurred in approximately 10 per cent, renal tuberculous lesions in 4 per cent, rectal tuberculous lesions in 3.5 per cent, lymphadenitis in 3 per cent, otitis media in 2.5 per cent, and peritonitis in 2 per cent. Tonsillar lesions were recorded in 6, skin lesions in 4. Multiple extrapulmonary lesions occurred in all groups and were as common with minimal as with far advanced pulmonary disease, also in 16 per cent of the childhood type. From the converse standpoint, Harris, of Toronto, has found definite pulmonary lesions in 60 per cent of orthopaedic and 62.5 per cent of renal cases. Renal tuberculosis has been found particularly prone to accompany skeletal lesions. Among the skeletal lesions in pulmonary tuberculosis, spinal lesions predominate, with 42 per cent, and spinal and sacroiliac lesions combined total 58 per cent.—*The Association of Intra and Extrathoracic Tuberculosis, C. K. Petter, Tubercle, October, 1937, xix, 28.*—(*A. P.*)

Azygos Lobe.—Twenty cases in which an azygos lobe was demonstrated by X-ray are reported. Of this group, the azygos lobe was located on the left side in 2 cases, the remainder being found on the right. Pathological changes such as pleuritis, atelectasis and pneumonia, were observed in the azygos lobe in 6 cases of the series.—*Zum Studium des überzähligen Lungenlappens (Lobus azygos), C. Bacanu, Ztschr. f. Tuberk., September, 1938, lxxx, 355.*—(*A. B. C.*)

Progressive Primary Complex.—In 1,200 postmortem examinations only one case of an active primary complex in an adult has been found. In this case there was found cavitation in a primary complex in the middle lobe of the right lung of a twenty-two year old Negro male. From this there resulted diffuse haematogenous and lymphatic dissemination of nodular caseous foci to the neighboring lymph nodes, the adrenals, liver, kidneys, spleen, intestines and meninges.—*Progressive Primary Complex in an Adult, S. H. Belgorod, Quart. Bull. Sea View Hosp., April, 1938, iii, 306.*—(*A. A. E.*)

Fatal Haemorrhage.—At Sea View Hospital fatal pulmonary haemorrhages occurred in only 41 out of 984 cases in whom necropsy was performed in the past five

years. In 32 of these cases, ruptured aneurysms of the pulmonary artery were found. The case now presented is that of a forty-six year old elevator operator who had been discharged nine years previously as an arrested case. During those nine years repeated examinations failed to reveal any signs of active tuberculosis. Then suddenly he had a single, large, fatal haemoptysis. At autopsy there was found extensive healed pulmonary tuberculosis. However, healing had not been complete since there was active tuberculous tissue in the neighborhood of a main branch of the pulmonary artery. This granulation tissue had invaded the wall of the artery and led to its subsequent rupture. —*An Unusual Form of Fatal Hemorrhage in the Course of Chronic Pulmonary Tuberculosis Clinically Arrested, E. Klosk, Quart. Bull. Sea View Hosp., January, 1938, iii, 176.*—(*A. A. E.*)

Alveolar Walls of Cat.—The silver lineation of the surface of the alveolar walls was produced by applying a weak solution of silver nitrate intratracheally and exposing to the sun or photographic developer. A network of silver lines is produced that can be traced through the main conducting division of the bronchial tree to the walls of the alveolar sacs of the respiratory division. In the smooth walled bronchioles the meshes of the network are small and fairly uniform. In the region of the respiratory bronchioles and alveolar ducts some of the meshes are larger, and, as one passes into the alveolar sacs, the number of large meshes increases while the smaller become relatively reduced in number. The smaller meshes form a continuous membrane, break up into rows or patches and small groups, and on the definitive alveolar walls they occur singly. The lines of the small meshes are continuous with the large meshes. In stained preparations, the small meshes are seen to represent the boundaries of epithelial cells lining the bronchioles, hence of endodermal origin, and can be traced to the alveolar walls where they have been given the name of epicytes. The large meshes represent the boundaries of bare areas in which no nuclei or other cell structure can be demonstrated. They probably correspond to the "nonnucleated squames" of earlier authors. They are more extensive where the capillary net is richest. It is thought probable that the bare areas form in association with the developing capillaries, possibly by the loss of epithelial cells lining the bronchioles as they expand to form alveoli. They do not appear to be projections or wings extending from the epicytes. If the bare areas retain none of their original protoplasmic covering they must then be made up of a differentiated ground substance or a modified basement membrane. If they are covered by an extremely thin nonnucleated protoplasm of the original epithelial cells it would seem to be composed mainly of water and can contain little or no stainable material. However, the fact that it cannot be demonstrated histologically does not prove it nonexistent. In these preparations pores are seen, frequently encircled by a silver line, and occasionally the lines on the surface of one alveolus may extend through the pores to be continuous with those of an adjacent alveolus. It would seem that occasionally the pores represent a place where an epicyte has been lost, much as a plug might be removed, forming an opening between adjacent alveoli.—*The Silver Lineation on the Surface of the Pulmonic Alveolar Walls of the Mature Cat, Produced by Applying Weak Silver Nitrate Solution and Exposing to Sunrays or Photographic Developer, C. C. Macklin, J. Thoracic Surg., June, 1938, vii, 536.*—(*L. F. B.*)

Bone Marrow Changes in Tuberculosis.—A study was made of the changes appearing in the bone marrow of 40 tuberculous patients, most of them in the advanced stages of the disease, the material for the study having been obtained *in vivo* by sternopuncture. In the majority of the cases the medullary elements appeared morphologically normal, the alterations most frequently observed consisting of anomalies in their development. The nucleus and the protoplasm of both the leukopoietic and erythropoietic elements showed different degrees of maturation, the first being usually more mature than the second. In some cells this dissociation was so marked as to make their classification difficult. Degenerative changes were also observed, such as single and multiple vacuoles in the protoplasm, and particularly toxic alterations of the granulations in the granulocytic series, entirely similar to those described by Naegeli in the peripheral blood. These last findings led the author to disagree with Naegeli's and Gloor's theory that such changes are of peripheral origin. For a quantitative comparison, the author used the myelograms established for normal individuals by Moisseieff and by Escudero and Varela. Although the total and relative ratios varied greatly in each individual case, he found that his cases on the whole presented a scarcity of nondifferentiated and immature elements, especially of megakaryoblasts and megakaryocytes, while presenting on the other hand an abundance of mature cells and accelerated curves of maturation. The karyokinetic quotas also appeared increased,

especially in the red series, in which mitosis was found in as many as 25 per cent of the cells. Correlation of these changes with the findings in the peripheral blood showed that on the whole medullary leukogenesis corresponded to a peripheral leukocytosis, while erythrogenesis corresponded to a more or less marked anaemia, and global medullary hyperactivity corresponded to a combination of anaemia and leukocytosis. The comparative and simultaneous analysis of the above findings showed that 95 per cent of the cases presented a medullary hyperactivity, which was global in 40 per cent, mostly erythrogenic in 32.5 per cent, and mostly leukogenetic in 22.5 per cent. In only 5 per cent of the cases the medullary activity appeared insufficient. The conclusions drawn by the author are that, from a physiopathological point of view, the medullary hyperactivity is to be considered as a response to increased stimuli from the periphery. On the other hand, the disproportion in single cases between the medullary hyperactivity and the peripheral needs, and particularly the degenerative changes observed in most cases, would suggest that an important rôle is played in these functional alterations by a myelotoxic factor, for which further studies should be considered.—*Ricerche morfologiche sul midollo osseo dei tubercolotici prelevato in vita mediante sternopuntura, G. Lanza, Riv. di pat. e clin. d. tuberc., January 31, 1938, xii, 1.*—(*G. W. W.*)

Intimal Changes in Branches of Portal Vein.—One-half cubic centimetre of a suspension of tubercle bacilli was inoculated into the right thigh of each of twelve 200 to 250 gram guinea pigs of both sexes. Five strains of organisms of varying virulence for guinea pigs were employed. Three of these strains had been recovered by the Loewenstein technique from patients suffering from hepatitis. Two animals died of tuberculosis five and six weeks after inoculation. Three died at from four and one-half to twenty-three hours following injection of tuberculin five to six weeks after the original inoculation. The others were chloroformed after varying periods of infection up to ten weeks. A careful study was made of the branches of the portal vein with particular reference to exudative changes of the intima. Particular attention was paid to vessels which were adjacent to or nearby specific tuberculous granulation tissue. The latter had not in most instances directly involved the vessel wall. Changes in the intima were occasionally observed in vessels surrounded for some distance by normal periportal connective tissue. The earliest changes noted involved the endothelium. The nuclei of the endothelial cells were no longer arranged in line. Single epithelial cells were raised up by the swollen subendothelium and their nuclei appeared to protrude into the lumen. Many of the nuclei were poor in chromatin. In other regions of the same vessel the nuclei appeared denser than usual and were occasionally swollen. Following this, fibrinoid changes appeared in the subendothelial layer; the elastic lamellae of the media were loose and frayed. The subendothelial cells contained swollen nuclei. In this layer appeared round and polygonal cells with a moderate amount of protoplasm. These cells were poorly delimited from the subendothelial ground substance. Their nuclei were round, occasionally rather long and large and somewhat chromatin-poor like lymphoid cells. These large mononuclear cells resembled to a certain extent the epithelioid cells which surrounded the vessel, but their nuclei stained more deeply and they were observed at times in the intima and even projecting into the lumen. Further changes were observed when the specific granulomatous process involved the vessel wall. At this stage many isolated young vessels were noted in the granulation tissue. The end stage appeared to be the development of hyaline connective tissue with more or less obliteration of the lumen. The cicatrization of the inner layers of the vessel walls was much more intense than that of the ordinary specific reaction seen in the periportal tissue. In only one instance could tubercle bacilli be demonstrated in the altered intima. Why the branches of the portal veins should be especially vulnerable is a matter for conjecture. They are thin walled vessels with no muscularis or elastica. In all probability bacilli pass through the vessel wall to the perivascular tissues and there an inflammatory reaction first occurs with granuloma formation. In one instance caseation was present. The intimal changes would thus appear to be perifocal reactions which for some unknown reason involve the intima first and the media only secondarily. At least no demonstrable changes are present in the media during the first stage of the process.—*Die Intimaveränderungen der intrahepatischen Pfortaderäste bei experimenteller Meerschweinchentuberkulose, F. Hausbrandt, Virchows Arch. f. path. Anat., January, 1938, ccci, 223.*—(*C. L. D.*)

Vascular Changes in Tuberculosis of Lung.—The lungs from six to eleven cases of each of several types of pulmonary tuberculosis were studied. Seven samples were taken from each specimen for microscopical study. The sections were stained with haematoxylin and eosin. Some were cut seri-

ally. Vascular changes were found to be both specific and nonspecific. Of the specific changes, tubercles were present in the vessel walls in cases with miliary spread. The tubercles were confined to the intima and the adventitia. In vessels lying in or near a tuberculous focus, in the epithelioid zone, or in the centre of a not yet caseated tubercle, specific granulomatous changes were observed. They consisted of epithelioid cells, a few lymphocytes and giant cells, with partial destruction of the vessel wall and of the elastic fibres. Where the adventitia had been infiltrated with epithelioid cells it could scarcely be distinguished from the surrounding tissue. The media was at times similarly involved, and the elastica interrupted by epithelioid cells. The endothelium might consist only of remnants having been overgrown by epithelioid cells which have filled the lumen of the vessel. Rarely did caseation involve the vessel wall, and caseous material spill into the lumen, for the latter was usually occluded before this could take place. The nonspecific changes were very striking, and consisted of a panarteritis. The adventitia was thickened and infiltrated with lymphocytes, a few polymorphonuclear leucocytes and spindle cells. The media was loosened and widened by lymphocytic infiltration. The number of elastic fibres was increased. Between the internal elastic layer and the endothelium there were great numbers of oval and spindle cells, with occasional lymphocytes and polymorphonuclear leucocytes. Deposit of fibrin was rarely seen. The endothelium was usually intact. In the area of caseation in primary foci, remnants of the elastica may be all that is left of the vessel, and the lumen filled with a necrotic mass. In the noncaseous islands of tissue the reaction was nonspecific, and the lumina were obliterated by cells. Many new blood-filled capillaries were present in the capsule proportional to the amount of connective tissue developed. Practically all of the vessels in the area of caseation were closed, fifty per cent of those in the capsule, and 10 per cent of those 1 to 2 mm. from the caseous area. Those 2 to 5 mm. away in air-containing lung tissue were quite normal. In regions of caseous pneumonia the changes were all nonspecific. The elastica was usually intact. Exceptions to this occurred chiefly in the veins. The lumina were obliterated by fibrin, and only rarely contained new capillaries. These capillaries which were large and dilated occurred only in the islands of less involved tissue. About 70 per cent of the vessels in the caseous areas were entirely closed, while twenty per cent were entirely open. In the nonspecific pneumonic area one mm. from the caseation, seventy per cent of the vessels were entirely open. In regions with bronchogenic spread the changes were specific. Only the elastica was left of the vessels in the centre of the focus. In the periphery of the central area of caseation the lumina of the vessels were closed by cells. In the epithelioid zone the vessels were closed by epithelioid cells, and in the lymphocytic zone by lymphocytes. Open capillaries were abundant only in the capsule. All of the vessels in the centre were closed and about forty per cent of those in the capsule. One-half to one mm. from the edge of the caseation in air-containing tissue the vessels were all normal. In the caseous centre of a productive focus the condition of the vessels varied with the age of the focus. The vessels were obliterated by the specific process, and the elastica was usually intact, except in instances where the focus was an old one. In the connective tissue zone the changes were nonspecific. Here slightly over one-third of the vessels were completely closed and an equal portion completely open. In miliary tubercles the vessels in the central part were closed by the specific process. Open blood-filled capillaries were frequently present in the capsule. The majority of the vessels in the caseous and noncaseous centres were closed. In the capsule slightly over fifty per cent of the vessels were closed, the majority by a nonspecific process. In the central caseous zone of tuberculous cavities the vessels were all closed by the specific process and filled with necrotic material. The elastica was mostly gone. In the capsule fifty per cent of the vessels were closed by a nonspecific process, with many capillaries present in the abundant connective tissue. The actual presence of caseous material in the vessel was uncommon. It was noted in fifteen large vessels most of which were veins. Diffuse intimal changes are uncommon, and no evidence of caseous thrombosis was seen. In the capsules of the lesions closed vessels were met with in the productive type of tuberculosis, bronchogenic spread, tuberculous cavitation, caseous pneumonia, miliary tuberculosis, and the primary focus in a descending order of frequency. In the caseous areas there was more vascular change in the presence of the exudative forms of tuberculosis, than in the presence of the productive forms, even though the caseous areas appeared to be morphologically similar. Nonspecific vascular changes were more frequently encountered in caseous pneumonias and primary foci. In all types there were vascular changes in from twenty to fifty per cent of the vessels in those areas with a nonspecific partly pneumonic and partly connective tissue reaction. This fact suggests that the

vascular changes precede the spread of the tuberculous process. This would decrease spread by the lymphatics and blood-stream. The accompanying formation of capillaries would tend to promote fibrosis about the focus. The specific and nonspecific vascular changes favoring the dissolution of tissue would also tend to favor the occurrence of caseous pneumonia.—*Die Gefässveränderungen bei den verschiedenen Formen der Lungentuberkulose, B. Neubert, Virchows Arch. f. path. Anat., January, 1938, ccci, 364.*—(*C. L. D.*)

Boeck's Sarcoid.—In this man symptoms began at forty-nine with generalized periarticular involvement, weight loss and cough. Roentgen examination disclosed huge bilateral symmetrically enlarged hilar lymph nodes. Ten months later, a roentgenogram showed complete disappearance of the enlarged hilar lymph nodes. Two years after the onset, the massive hilar shadows were again visualized. The patient died two and one-half years after onset of symptoms. At autopsy the tracheobronchial lymph nodes were the seat of extensive sarcoid involvement; they were markedly oedematous. This oedema probably explains the variation in the size of these nodes during life. Typical sarcoid lesions were present in the trachea, lungs, skin, myocardium, abdominal lymph nodes, liver, spleen, thyroid and kidney. In the latter two organs sarcoid lesions are here described for the first time.—*Boeck's Sarcoid, J. Spencer & S. Warren, Arch. Int. Med., August, 1938, lxii, 285.*—(*H. R. N.*)

Tuberculous Infection in Guinea Pigs.—In the present study the reactions produced by massive intravenous injections of avirulent and virulent human bacilli are compared. The author points out that in the recorded findings of other investigators the suspensions used for injection were fairly coarse and "all interpretations based on the use of such suspensions must be evaluated carefully with this in mind." However, a study of the text reveals that the main difficulty encountered in the present experiment was the inability of suspending as much as 100 mg. without lumps of considerable size relatively, since the volumes of liquid used in the guinea pig could not be excessive (10 to 20 cc.). The tissue responses to the intravenous injection (ear vein) of large amounts (100 mg.) of avirulent human tubercle bacilli (in as fine a suspension as can be prepared feasibly for this purpose) are those characterized by Opie as resembling closely the changes that occur at the onset of inflammation produced by a great variety of irritants, namely, exudative and proliferative reactions, the gradation of which is affected in various places in the tissues by the numbers of bacilli lodging there. With these large amounts, the greater part of the lumps lodge in the lungs, and the histiocytic reaction in the lungs is more pronounced than that observed in the liver, spleen or kidney. With these large amounts, the cellular and tissue resolution closely approximates that of the presence of viable bacilli (determined by culture) in the tissues and is correlated with the approximate numbers required to cause a discernible tissue response. For quite some time before tissue resolution has occurred, acid-fast bacilli are no longer to be seen and, for some time after acid-fast bacilli are absent, acid-fast globules and granules are noted in the tissues. No evidence of multiplication of avirulent human or bovine (BCG) bacilli was noted even after large amounts (from 100 to 200 mg.) were injected; their "toxicity" in these large amounts was variable and dependent on the culture medium used for growing them. Virulent human tubercle bacilli when given in equally large amounts intravenously caused tissue reactions which did not differ strikingly except in the grade of reaction, especially after the first week. However, distinct evidence of increase in the number of bacilli in the tissues was found, correlating with cultural findings. It appears that in normal guinea pigs the tissue responses to large amounts of avirulent bacilli intravenously injected are influenced by the local number and disposition of the bacilli. On the other hand, the tissue reaction to virulent bacilli is a response to numbers rapidly increasing locally and distributed through the circulation.—*Course of Experimental Tuberculous Infection in Guinea Pigs. Pathological Changes Produced by Massive Intravenous Injections of Virulent and Avirulent Tubercle Bacilli, H. J. Corper, Arch. Path., July, 1938, xxvi, 109.*—(*J. S. W.*)

Spontaneous Tuberculosis of Guinea Pigs.—The existence of spontaneous tuberculosis in guinea pigs has been demonstrated by various investigators to be an indisputable fact and to occur with relative frequency. Cohabitation of new guinea pigs with infected ones is sufficient to cause tuberculosis in the animals in periods varying from six to twenty months. Tuberculosis contracted in this manner is of the aerogenous type. The lesions moreover resemble those observed in human tuberculosis. The lesions of spontaneous tuberculosis of 21 guinea pigs observed were found to be very like those of human tuberculosis. The initial lesion was almost always single, intrapulmonary

and with the right lung more often involved than the left. Mediastinal adenopathy was marked and bilateral. In all the cases observed a generalized tuberculosis had developed. It was clear that the bronchial route, as in man, had played a certain rôle. From a diagnostic point of view, there should be no confusion of spontaneous tuberculosis with an inoculation infection. In the former the initial pulmonary lesion is large, with marked mediastinal adenopathy, whereas in the latter the lesion is inguinal, at the site of inoculation. Also the rapidity of the infection by inoculation and the slow development of spontaneous tuberculosis permits a differential diagnosis. The fact that the form of spontaneous tuberculosis in guinea pigs approaches the human type of tuberculosis should not be overlooked.—*Sur la similitude des lésions de primo-infection pulmonaire dans la tuberculose humaine et dans la tuberculose spontanée du cobave, A. Saenz & G. Cannetti, Compt. rend. Soc. de biol., February 26, 1938, cxxvii, 661.*—(*M. T. W.*)

Vital Capacity.—According to various clinical and experimental observations the amount of air within the lungs is, in general, inversely proportional to the amount of circulating blood within the lesser circulation. The determination of the vital capacity is a useful method of measuring the air content of the lungs, when there are no complicating factors such as disease of the lungs or the thoracic cage. The vital capacity is diminished in cardiac patients with acute or chronic pulmonary congestion. Phlebotomy of from 500 to 1,000 cc. in normals leads to a definite increase in vital capacity. Ten normal students were employed for observation. Tourniquets were applied to all four extremities so as to obstruct only the venous flow. In every case, after a few minutes, there was an increase in vital capacity, from 300 to 850 cc. After removal of the tourniquets the vital capacity shortly returned to normal. There was a definite rise in vital capacity when venous compression was applied only to the lower extremities; there was a further, but proportionately smaller, increase when compression was also applied to the upper extremities. The effect on the vital capacity can only be explained by the diminution of the pulmonary blood volume resulting from the obstruction to the venous outflow from the extremities. It is well known that the vital capacity is greater in the erect position than in the horizontal, particularly under conditions of minimal muscular exertion. This is most probably explained by the lower venous pressure, diminished cardiac output and consequent diminished pulmonary blood volume. In a series of subjects, in the erect position, the vital capacity constantly rose while this position was maintained; some of the subjects maintained a stationary erect position until they were close to collapse. The application of tourniquets to subjects in the sitting or standing position results in an increase of vital capacity, but this is definitely less marked than when the subject is lying down. These observations can be applied clinically in such conditions as acute left ventricular failure and acute pulmonary oedema. In these cases the immersion of the extremities in warm water or the application of tourniquets obstructing the venous outflow (bloodless phlebotomy) may prove of definite value.—*Zur Beeinflussung der Vitalkapazität der Lunge durch Behinderung des venösen Abflusses aus den Extremitäten (Stauung), G. Budelmann, Klin. Wchnschr., July 16, 1938, xvii, 1009.*—(*H. R. N.*)

Pulmonary Function.—The intrinsic functions of the lungs are their alveo-capillary surface-creating and self-cleansing powers. Disturbance in these functions becomes evident by obliteration of some areas of the lung and compensatory hyperfunction of others. In pulmonary fibrosis there is a permanent loss of functioning units and in emphysema there occurs a loss of retractive power, failure to provide new breathing surfaces and consequent overstretching of the available spaces. In the lungs there is a specific function analagous in every sense to the special functions of other organs; and, as in other organs, the lungs are composed of units with capacity for independence of function and potentialities for compensatory hyperfunction in case of failure of other units. The characteristic structural units of the lungs are the air spaces; these units function in alternation when the body is at rest. The intrinsic function of the pulmonary units is that of adaptation of their structures to create an adequate surface for the exposure of the blood to meet the requirements of the body for gas exchange and evaporation. The antenatal development of the lungs takes place by growth of the primitive anlagen of entoderm and mesoderm. The first air spaces are produced by the mechanical disruption of the continuity of the entodermal lining. At birth the centrifugal thoracic force (the action of the muscles of respiration) and the centripetal circulatory force (the erectile action of the pulmonary capillaries) act together to disrupt the entodermal lining of the units, to expand these into air spaces, and thereby expose the capillary network which thus becomes separated from the outside air only by the

delicate mesenchyme. These functional mechanisms continue through life. Postnatal growth and development of function keep pace with increasing demand. In function there are definite consistency changes in the mesenchyme matrix as well as in the endothelial membrane of the capillaries; it is these changes which, in part, allow for the expansion of the air spaces and the protrusion of the capillaries into the air spaces by means of their centripetal erectile force. In functioning in man the air spaces are at times lined by epithelium and at other times the capillaries are entirely exposed; resting units are fully lined, their walls are thickened and they have the appearance of "physiological atelectasis." Functioning units are expanded, their walls are thin, and their epithelial linings are discontinuous: thus, there are naked stretches where the capillary surface is exposed with only an extremely thin mesenchymal membrane between it and the air space. In the resting unit, blood is supplied by the bronchial arteries by anastomoses with the capillary net of the air spaces derived from the pulmonary artery. Increased capillarization is the key to increased function. Increased unit function affords self-cleansing of the air spaces and of the breathing surface. Intrinsic unit function and ventilation (extrinsic breathing) are closely correlated. The former is uninterrupted but operates in alternating groups of units; but the latter is periodic depending upon the rhythmic contractions and relaxations of the neuromuscular apparatus of breathing. The ventilatory equipment, while chiefly under voluntary control, is subservient to intrinsic lung function which is under vegetative nervous control. With the demand for increased function, as in exercise, there is an increase in unit function and in the number of functioning units, and, with this, ventilation increases proportionately. Interference with normal variations in the consistency of the mesenchymal matrix and of the capillary endothelial membrane leads to hardening of the matrix; as a result there is diminution in mobility and permeability. The function of self-cleansing is thereby impaired; fibrosis and obliteration of the affected units are the result. However, the expansile force of thoracic traction still acts upon the pulmonary unit despite the fact that it has been excluded from intrinsic function. The result is emphysema which is the expansion of the unit without simultaneous creation of breathing surface. Fibrosis and emphysema are always concomitant and the net result is determined by the quantitative changes. Clinically, emphysema means the permanent distention of the lungs to a degree which normally prevails transitorily in the increased function occurring during exercise. The permanent state of expansion depends upon the basic tonus of the neuromuscular apparatus of breathing; and the periodic excursions of breathing depend upon the rhythmic contractions of this same apparatus. Hence, if the basic tonus must increase to hold the lungs in greater permanent expansion, then the periodic contractions must diminish in amplitude; in short, ventilation becomes less efficient. As fibrosis obliterates an increasing number of lung units, more units are called upon for replacement; this increases the permanent expansion of the lung which ultimately leads to emphysema with restriction of vital capacity. When fibrosis leads to obliteration of so many units that the requisite number of units is no longer available, the force of thoracic traction keeps the still available units in a state of permanent expansion. The remaining units may then become thinned out and bullous. Emphysema represents air-space production without concomitant breathing-surface creation. Just as fibrosis represents failure in the function of self-cleansing, so emphysema represents failure in the function of breathing-surface creation. As fibrosis and emphysema become more advanced we observe episodes of pulmonary decompensation manifested by dyspnoea, cyanosis and pulmonary oedema. Finally all cases arrive at the stage of acute pulmonary oedema which has been interpreted as cardiac failure. However, it is not the heart which fails but rather its increasing compensatory effort which intensifies pulmonary embarrassment. With increasing numbers of units out of function, the circulation must increase in the remaining available units; the blood continuing to come into the lungs only increases the burden of the few remaining units and these eventually fail in their function of self-cleansing and breathing-surface creation. The result is the death of the patient with pulmonary oedema due to acute failure of function of the pulmonary units.—*The Relation of Pulmonary Function to Fibrosis and Emphysema, J. A. Miller & I. Rappaport, Ann. Int. Med., March, 1938, xi, 1644.* —(*A. A. E.*)

Circulation Time.—The circulatory pathway measured in this study is that from the antecubital vein to the pulmonary capillaries. This has been designated the "pulmonary circulation." The method used was the injection of 1.4 cc. of paraldehyde into the antecubital vein; this drug, thus administered, regularly causes coughing. The period of time between injection of the

paraldehyde and the appearance of the cough was measured with a stopwatch. Tests were made on one hundred males and females from fifteen to seventy years of age, and all with apparently normal cardiovascular systems. The average "pulmonary circulation time" was found to be 6 seconds with a normal range of from 3 to 9.5 seconds. The cough which was produced lasted from one to three minutes and was usually of moderate severity.—*Determination of the Normal Circulation Time from the Antecubital Veins to the Pulmonary Capillaries by a New Technic, S. Caud, Ann. Int. Med., August, 1938, xii, 236.*—(*A. A. E.*)

Variations in Volume and Circulation of Lung.—Under physiological conditions the volume of the viscera is not stable. All the organs are capable of changing their volume either by mechanical or reflex action. Congestion of an organ, whether inflammatory or hyperfunctional in origin, increases the volume, whereas certain pharmacodynamic substances lessen it. Modifications in volume are greater in elastic organs, chiefly the lungs. The volume of the lung is modified not only mechanically during the act of respiration but also by neurogenic activity. It might seem logical to make a distinction between the causes that provoke the variation in the volume of organs, whether mechanical or neurogenic; scientifically, such a distinction is not tenable for these two factors are interdependent. Actually, the mechanical action is governed to a certain degree by nervous action and vice versa. The modifications of the volume of the viscera of nervous (reflex) origin are produced by stimulation. These reflexes fall into two categories, viscero-visceral and cutaneous-visceral. The viscero-visceral reflexes occur by direct stimulation on the pleura or on the nervous ganglia in the thorax, in the lung or in other organs resulting in changes in pulmonary volume. The following experimental evidence substantiates the above. If one stimulates the ends of the vagus nerve, distension of the lung results (Rosenthal, Schepelmann, etc.). Second, if one stimulates either directly or indirectly the pleural reflexes or the parts adjoining the posterior mediastinum and hilum, distension of the lung results (Rosenthal, Walker, etc.). Third, stimulation of a superior cervical ganglion in a dog produces sudden distension of the lung; resection of the superior cervical sympathetic produces permanent pulmonary dilatation with an enlargement of the hemithorax (Traube, Rosenbach, etc.). Clinically, there are evidences of these pleural reflexes. Pleural irritation following the introduction of air or different liquids (hypertonic saline, gomenol oil) can produce alveolar atelectasis or even an overdistension in the different lobes. This often leads to an apparent disappearance of a cavity or a modification of the pulmonary circulation with the formation of a small pleural effusion. Also, variations in abdominal pressure, such as produced by digestion, influence the volume of the lung. Artificial pneumoperitoneum will produce the same effect. Sometimes, during the course of a pneumothorax one sees an unexpected rapid and persistent complete collapse of the upper lobe. Certain pulmonary cavities vary considerably in size from time to time, often disappearing radiographically within a few days. This reduction in the size of the cavity is due to active lobar contraction of reflex origin. As a result of his experiments the author feels that certain general conclusions of fundamental value can be stated as follows: (*1*) the lung is an organ with a definite tone which can be automatically modified by reflex action and by artificial or natural stimulation applied both through the viscera and the skin; (*2*) these modifications in tone, when caused by stimulations applied directly to the pleura or to the lung, are accompanied by variations in pulmonary volume in the nature of expansion or contraction, varying in degree with the individual; (*3*) certain stimulations carried to the pleura, to the lung, to the visual apparatus and to certain mucous membranes are capable of exciting vasomotor changes in the lung, that is, vaso-constriction or vasodilation, varying with the subject; (*4*) a large number of individuals respond to stimulations which modify the volume of an organ and which excite the vasomotor centre; (*5*) psychical (emotional) stimulations such as fear are capable of producing variations in pulmonary circulation. It is probable that not only the problem of recovery, but also the solution of the problem of the pathogenesis of tuberculosis ought to be investigated in the light of primary functional disturbances of the pulmonary tissue of neuro-reflex origin. In the treatment of tuberculosis it is necessary in all cases to fix attention more on the biological factors than on mechanical factors.— *Variation du volume et de la circulation du poumon d'origine réflexe, F. Parodi, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 1, 8.*—(*J. E. F.*)

Cardiopulmonary Function Test.—Two stages of heart failure can be distinguished: the latent stage, during which the patient in the resting condition does not show any signs of insufficiency, but is more or less incapable of physical exertion owing

to dyspnoea; and the stage of decompensation in which are found the typical symptoms of insufficiency. In cases of pronounced congestive heart failure, there is a marked increase in standard metabolism; after successful treatment, the condition of these patients may be characterized as that of the latent stage. Simultaneously, there is a decrease of the standard metabolism to normal values. As dyspnoea on exertion is the most important symptom of latent cardiac insufficiency, it is essential to devise a method whereby this symptom may be gauged. The author has based his technique on the evidence of increased oxygen consumption in the normal after hard work, called "oxygen debt," after Hill. It has been shown that the decompensated patient manifests a higher oxygen debt, after graduated work, than the normal person. The author's apparatus consists of four sections of stairs, each, one metre in height and forming a circle when put together. Each section consists of six steps. There are three degrees of work. The lightest test (5/88) consists of five rounds on the stairs at a pace, set by a metronome, of 88 steps per minute, a performance which most patients, even with severe insufficiency, are able to achieve. The moderately strenuous test (5/160) consists of five rounds at 160 steps per minute. The strenuous function test (10/208) consists of ten rounds at a rate of 208 steps per minute. The last test is useful in demonstrating latent cardiac insufficiency. The oxygen consumption is determined by a Krogh spirometer. A metabolism curve is first made under basal conditions. With the subject in a semi-recumbent position, the oxygen consumption is then determined exactly from the beginning of the second minute to the end of the fifth minute after the performance of work on the stairs. The increase in oxygen consumption after work per minute, "relative oxygen debt," is then calculated as a percentage of the resting value. In 265 normal persons the upper limit with the light test was 29 per cent, with the moderately strenuous 76 per cent, and with the strenuous test 114 per cent. The relative oxygen debt for healthy individuals is independent of body weight, provided the latter is physiological. When the body weight is excessive, the relative oxygen debt is abnormally high. Reduction of weight in these cases brings the oxygen debt back to normal. In severe cardiac insufficiency high values of the relative oxygen debt are observed; with the moderate test (5/160) values up to 150 to 200 per cent were seen. In patients with valvular affections, essential hypertension and coronary artery disease, with no apparent symptoms, the stair test is useful in determining cardiac reserve. The test aids particularly in discovering cases with latent cardiac insufficiency. Illustrative cases are cited. In pulmonary diseases some individuals tolerate a considerable, others a slight reduction in vital capacity without dyspnoea on exertion. Estimation of the relative oxygen debt in these patients permits, in some degree, determination of their performance capacity. Using the author's test Blum measured the work dyspnoea of patients with pulmonary tuberculosis with and without pneumothorax, and found the test more informative in measuring work tolerance than determination of vital capacity. In two cases the vital capacity was reduced to half of normal by a spontaneous pneumothorax, and by compressing the chest with a girdle; the relative oxygen debt remained at normal value under the most strenuous test. Many forms of silicosis do not give rise to definite insufficiency symptoms by this test until the lesion is far advanced. Both heart and lung disease may be present in the same case, with marked reduction in vital capacity, without appreciable reduction in capacity for work as determined by the stair test.—*The Practical Applicability of the Cardio-Pulmonary Function Test, G. Nylin, Acta med. Scandinav., Supplementum 93, 1938.*—(*H. R. N.*)

Cardiac Output.—A series of experiments was undertaken on dogs to study the effect of cardiac compression and compression of the intrathoracic pulmonary vessels, by continuous and intermittent inflation of the lungs, upon cardiac output. In four experiments the cardiac output during natural breathing was compared with that when the lungs were inflated at pressure from 20 to 30 mm. of mercury. In three, cardiac output showed a pronounced decrease, and in the fourth, the decrease was significant but not great. In seven experiments cardiac output was determined during natural breathing with the chest closed and during several periods of continuous inflation with the chest widely open. In five of the seven, cardiac output was decreased during the period in which the lungs did not fill the chest. In the other two, cardiac output increased, one slightly and the other pronouncedly. When the lungs were inflated to just fill the chest, cardiac output dropped to a lower level in two instances and was a little higher in one case. During the period of high intratracheal pressure with the lungs protruding from the chest, cardiac output was still lower. In nine experiments, cardiac output

during a control period was compared with that occurring when respiratory paralysis was produced with curare and the lungs were intermittently and continuously inflated. In six of eight cases, where curarization was followed by intermittent inflation, cardiac output decreased. In two others, there was both a slight rise and fall, respectively. With continuous inflation at a lower pressure, there was a rise in cardiac output in four instances, exceeding the control level. The experiments give some suggestion of the mechanism by which positive inflation may have a harmful effect on the circulation. They suggest that the danger of inflating the lungs, with the chest closed or open, is primarily due to a compression effect on the heart which interferes with its filling and not to pressure on the intrapulmonary vessels which creates a resistance against which the right heart must empty. It is important for an anaesthetist to realize that the lowest intratracheal pressure that will maintain ventilation is the safest, since there is no means of recording the compression effect of the lungs.—*Studies of the Cardiac Output of Anesthetized Dogs during Continuous and Intermittent Inflation of the Lungs, G. H. Humphreys, R. L. Moore, H. C. Maier & Virginia Apgar, J. Thoracic Surg., April, 1938, vii, 438.*—(*L. F. B.*)

Dual Pulmonary Circulation.—Using a 20 per cent suspension of bismuth oxychloride in 11 per cent acacia, the pulmonary arteries and bronchial arteries were injected in 74 cases that had died of various causes. Because of the viscosity of the medium, the injection material did not extend beyond vessels smaller than 12 to 15 micra in diameter. The groups of cases sufficiently large to justify conclusions were tuberculosis, cardiac disease and malignancies. It was found that changes in the bronchial arterial circulation occurred and were generally characteristic in various diseases. In primary malignancies there was a marked increase in the supply of bronchial arterial blood to the region involved. No changes appeared in cases of metastatic malignancies. It is likely that bleeding in cases of pulmonary neoplasm indicates primary and not metastatic tumor. In cases of old rheumatic heart disease, the bronchial arteries were slightly to moderately dilated and there was a constant widening of capillary anastomoses between the bronchial arteries and branches of the pulmonary arteries so that the injection medium poured into the branches of the pulmonary arteries. This finding was constant in cases of hypertrophy of the right ventricle and failure of the right heart. Similar changes, but less constant and in milder degree, were present in syphilitic heart disease, hypertension and generalized arteriosclerosis. In such cases there is clinical evidence indicative of increased pulmonary venous pressure. In 4 cases of coronary sclerosis no alteration of the bronchial circulation was found. In one case having extensive sclerosis of the pulmonary arteries, injection of the bronchial arteries culminated in an injection of the pulmonary arterial system. In every case of tuberculosis consistent changes in the bronchial arteries were found. The more chronic and extensive the tuberculosis, the more marked was the dilatation, tortuosity, and the more numerous the ramifications of the bronchial arteries. The studies indicate an increased supply of oxygenated blood about the diseased area. The dilatation of the bronchial arteries occurs in part as a result of obliteration of the pulmonary arterial bed in the diseased areas. In cases of bronchiectasis, lung abscess with bronchial fistula, polyserositis, chronic empyema and chronic lung abscesses there was dilatation of the smaller branches of the bronchial arteries associated with the establishment of many new vessels. In 2 cases each of asthma and emphysema, the finer branches of the bronchial arteries seemed more numerous and extended further into the parenchyma than normal. In congenital cystic disease there was slight to moderate dilatation of the bronchial arteries and a few bronchial-pulmonary artery anastomoses. In one case injection of the bronchial arteries readily filled the pulmonary arterial tree. The pulmonary arteries were displaced around the cysts.—*The Rôle of the Dual Pulmonary Circulation in Various Pathologic Conditions of the Lungs, D. A. Wood & Miriam Miller, J. Thoracic Surg., August, 1938, vii, 649.*—(*L. F. B.*)

Dyspnoea.—The causes of dyspnoea are reviewed and discussed. Despite the multiplicity of clinical conditions in which dyspnoea is observed, the fundamental mechanisms of its production are either chemical or reflex disturbances. Acidosis and anoxaemia constitute the chemical disturbances and account only for the dyspnoea of emphysema, acid poisoning and diabetic acidosis. Reflex disturbances, mainly from the "stretch receptors" in the lungs, however, play a more important rôle and are associated with pulmonary congestion. On the basis of reflex disturbances, therefore, the dyspnoea of lobar pneumonia, cardiac asthma and pneumothorax can be satisfactorily explained. To treat rationally conditions in which there is respiratory embarrassment, an understanding of these

fundamental mechanisms is required.—*Dyspnea: Review, R. V. Christie, Quart. J. Med., July, 1938, vii, 421.—(A. B. C.)*

Mechanism of Coughing.—The author has studied the mechanism of coughing by kymographic methods. Coughing is a sudden and forced movement of expiration whereby the normal rhythm and normal continuity of respiratory movements are interrupted voluntarily or involuntarily as a reflex of protection. Three stages may be distinguished during the act of coughing: first, a more or less deep inspiration precedes the coughing; second, a hacking respiratory movement during which the vocal cords are contracted; third, opening and sudden relaxation of the vocal cords, thus expelling the contents of the windpipe. Roentgen-kymographic studies of the act of coughing show that speed and amount of the fluctuations of the air volume occurring during the act of respiration and coughing are in direct ratio to the speed and size of the kymographic variations. During the act of coughing the respiratory mechanism of the lower pulmonary lobes in coördination with the mechanism of abdominal pressure plays the main physiological part. The diaphragmatic movement is the decisive factor in the act and effect of coughing. After phrenic nerve interruption the homolateral side has lost the ability to eliminate by coughing. There is evidently a process of suction going on from the disabled lung to the opposite lung. This might explain the increased expectoration which follows phrenic nerve interruption. There is also increased danger of aspiration of material from the diseased lung by the healthy side.—*Physio- und Pathomechanik des Hustenaktes, H. H. Weber, Radiologische Rundschau, May, 1938, vi, 135.—(F. G. K.)*

Pressures in Oesophagus, Stomach and Duodenum.—Measurements were made by having the patient swallow an ordinary Levine tube which was then connected to a water manometer. In 16 individuals the pressures in the oesophagus were negative becoming more so on inspiration and relatively more positive on expiration. In 23 individuals studied, the gastric pressures were found to be positive with occasional increase in pressure during inspiration; with any increase in abdominal tension, the pressures became more highly positive. In the duodenum the pressures were all positive. The pressures within the various organs are maintained by the action of the sphincters. From other experiments it is felt that the opening of the sphincters depends upon intragastric pressure. It appears that the sphincters are actually forced open by the increasing pressure rather than that they relax. The negative pressure in the oesophagus probably explains the aspiration of bronchial secretions or iodized oil from the trachea into the stomach.—*Some Observations on Pressures in the Oesophagus, Stomach and Duodenum and the Effect of these Pressures on the Opening and Closing of the Sphincters, G. G. Ornstein & D. Leibovici, Quart. Bull. Sea View Hosp., April, 1938, iii, 238.—(A. A. E.)*

Nitrogen Metabolism in Lung.—The authors have previously drawn attention to the fact that the lung exerts an influence on nitrogen metabolism. The lung is capable of splitting the polypeptides of the blood, resulting in an elevation of the amino-nitrogen in the blood. Also, the lung can act upon the amino-acids of the blood to liberate ammonia. Similar investigations, using the technique of the isolated, ventilated and perfused lung of a dog, were pursued. Study was directed to the action of the lungs on peptones and on the amino-acid arginine. The authors found that the perfused blood is enriched in polypeptide nitrogen while circulating through a lung scalded by hot water. This increase in polypeptide nitrogen diminishes if the lung is subsequently perfused with the blood of a normal lung. The perfusion experiments also showed that the lung is capable of attacking both peptones and the diamine-acid complex arginine. The latter is evidently acted on by an enzyme arginase, with urea as a chief product of the decomposition. It is concluded that the lung is capable of acting on nitrogenous materials just as it acts on fats and carbohydrates.—*Poumon et métabolisme azoté, L. Binet & M. Burstein, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 1, 1.—(J. E. F.)*

Bronchogenic Distribution of Fluid and Particulate Matter.—Bronchogenic distribution of particulate matter is generally said to affect the base of the lungs by preference. Nevertheless the spread of pulmonary tuberculosis, which in man takes place largely by the bronchial route, extends in the classic craniocaudal progression. In translateral extension the lesions are found in the cranial rather than in the caudal parts of the lung. In 102 routine autopsies in cases of pulmonary tuberculosis, pneumonoconiosis or a combination of the two diseases, there were 50 instances where the lesions were confined to restricted areas of obvious predomination. In over half the latter group the diseased area was observed as a transverse band which occupied on the

right the lower third of the upper lobe and the apex of the lower lobe, and on the left the middle third of the upper lobe and the apex of the lower lobe. There is therefore an obvious predilection for a part of the lung which will be termed the transverse band area. In lungs in which this area alone is affected the outline is sharp, and the shape is often somewhat conical, with the apex near the hilum and the base reaching into the peripheral parenchyma or even to the pleura. The configuration of this lesion could be exactly reproduced in the upper lobes of human lungs removed at autopsy by injecting an opaque substance into the bronchi reaching the transverse band areas. As further proof that the lesions in question are the result of bronchogenic spread, guinea pigs and rabbits into whose tracheas small amounts of india ink were injected showed deposition of the pigment in the transverse band area either exclusively or preponderantly. There is no conceivable haematogenous mechanism by which this lesion could be produced, particularly bilaterally. The mechanism of the selection of this special route for bronchogenic spread is demonstrable only with difficulty. Gravity alone is not a sufficient cause for the phenomenon. No matter what the position of the animal, the carbon is deposited in some part of the transverse band area. The dorsal or membranous portion of the bronchial tree is not flat as commonly taught, but shows two lateral grooves separated by a raised longitudinal fold. At the carina the fold forks and passes down each main bronchus together with the grooves. This process is repeated at each bronchial ostium. In the bronchi, however, the external lateral groove is broader and deeper than the internal lateral. Moreover, the external groove of each bronchus passed uninterruptedly into the external lateral groove of the most lateral bronchial branch whereas all the other grooves and bronchi are interrupted by the central elevated fold. Thus there is a system of lateral grooves continuous with the two lateral grooves of the trachea. Cross sections of the trachea, carina and secondary bronchi in the rabbits injected with india ink showed that the ink proceeds by way of the external grooves into the lateral and dorsal branches of both main bronchi and so on at each branching. This lateral shunt insures the development of a transverse band area in the lower portion of the upper lobes and the upper portions of the lower lobes. As has been mentioned, the transverse band area is observed not only in tuberculosis, but also in pneumonoconiosis. The involvement of this area has also been observed at autopsy in cases of lipoid pneumonia and of haemorrhage into the respiratory tract. The mechanism in question holds only for fluids or particulate matter suspended in fluid. Particulate matter suspended in air is not influenced by the architecture of the tracheobronchial walls. It shows axial flow in the column of respired air and is distributed in a uniform fashion to all parts of the lung. This mechanism would on the face of it contradict the observation made in cases of pneumonoconiosis. The presence of a definite transverse band in pneumonoconiosis is accounted for by the hypothesis that, despite the tendency to general distribution, some of the dust remains in the air passages, and, becoming caught in the mucus of the bronchial wall, is started on its way to the trachea by the cilia and the peristalsis of the bronchial musculature. In the latter instance the particulate matter is suspended in a fluid medium and is governed by the law of lateral flow which has been outlined. Hence the transverse band area gets more dust than the rest of the lung. It is quite possible that apical tuberculosis owes its prevalence in part to the lateral flow mechanism of transportation of particulate matter suspended in fluid.—*Bronchiogenic Distribution of Fluid and Particulate Matter. Its Site of Predilection and the Mechanism of Transfer, H. S. Reichle, Arch. Path., June, 1938, xxv, 811.*—(*J. S. W.*)

Experimental Tuberculous Panophthalmitis.—One-hundredth of a milligram of a suspension of tubercle bacilli was injected into the posterior chamber of the eyes of 28 rabbits. After injection an inflammatory reaction was always noted at the site of inoculation. This reaction varied in degree according to the virulence of the strain used and was least marked with strain BCG. The cellular spread was by contiguity. Infection of the vitreous humour did not always occur; sometimes the anterior segment of the eye was alone infected. When the posterior chamber was infected the extent of the lesion at the optic disc equalled in degree and intensity that at the site of inoculation. This could be explained in part by the anatomical structure of the vitreous humour, and in part by the main lymph drainage path posteriorly along the hyaloid canal. Generalized tuberculosis was found in 6 of the 13 rabbits inoculated with virulent strains, while none of the 15 rabbits inoculated with avirulent strains developed generalized tuberculosis.—*Experimental Tuberculosis of the Eye, W. A. Gray, J. Path. & Bact., November, 1937, xlv, 647.*—(*H. J. H.*)

Spread of Dye in Skin of Tuberculous Guinea Pigs.—The present study illustrates the use of a dye to determine the presence of the property of restriction of spread in the skin of allergic guinea pigs. The dye employed was pontamine sky blue, dialyzed in running water until free from salts, and then made into a 2.5 per cent solution (approximately isotonic). This was injected just beneath the epidermis in groups of normal and tuberculous guinea pigs, also in a few guinea pigs suffering from epizootic lymphadenitis and sensitive to a bacterial extract of the organism (streptococcus) responsible for this disease. The amount of the dye solution injected was as close to 0.025 cc. as possible. An electric animal clipper was found satisfactory for removing the hair. In general the injections were limited to either the dorsal or ventral zones. When the dorsal zone was used, two injections were made in the midline about 3 or 4 cm. apart; when the ventral zone was employed, four injections were made, one into each of the four quadrants. The areas of the spread of the dye were measured after one, four and twenty-four hours (where possible). The allergic tuberculous animals showed definitely much smaller areas of spread than the normals at every observation. Furthermore, in the normal skin the edge of the area showing dye was always hazy, while in the allergic tuberculous animal the corresponding area was sharp as a knife's edge. In fact, in many of the normals after twenty-four hours it was difficult to determine the outline of the area of primary injections, because the entire animal was stained, even the foot-pads, the tongue and the conjunctiva. It was found that there was no significant difference in the rate of spread of dye between guinea pigs that showed two-plus and four-plus tuberculin reactions. However, in the group of tuberculous guinea pigs with far advanced disease and with little or no skin response to tuberculin (anergic), the dye spread faster than in the normal animal. The 3 guinea pigs with epizootic lymphadenitis showed a significant restriction of the dye, but not as great as in tuberculous animals. It is clear from these experiments that at least two different processes are involved in this restriction to the spread of dye. They are a change in the rate of diffusion in the spaces of the connective tissues, and a decrease in the permeability of the endothelium of the lymphatic capillaries. The difference of drainage through the lymphatics into the blood-stream is especially evidenced by the fact that when as much as 0.1 cc. of dye solution was injected, as in the four injections in the ventral region, the dye had spread throughout the normal animal in twenty-four hours sufficient to stain all the mucous membranes, whereas in the tuberculous animal not enough had drained into the blood vessels to be seen away from the place of injection. Thus one factor, active in the allergic state, has to do with the permeability of endothelium. Infection with tuberculosis in guinea pigs induces changes which are manifest as an altering of the rate of diffusion in the connective tissues and of the rate of absorption through the vessels. These changes can be demonstrated not only by specific antigens but also by nonspecific materials.—*Altered Cutaneous Conditions in the Skin of Tuberculous Guinea Pigs as Demonstrated with a Vital Dye, (with one plate), A. L. Joyner & F. R. Sabin, J. Exper. Med., September 1, 1938, lxviii, 325.—(J. S. W.)*

Leucocytic Response in Sensitized Calves.—In a previous experiment an attempt was made to produce a leucocytic response in rabbits comparable to the leukemoid reaction seen in human tuberculosis. A striking elevation in the leucocyte count with a marked shift to the left of the granulocytic cells occurred following the injection of tuberculin in rabbits previously infected with tuberculosis. Since rabbits are very sensitive to most haematopoietic stimuli, it seemed desirable to determine whether similar reactions could be induced in other animals. Therefore tuberculin-negative calves were infected with a virulent strain of bovine tubercle bacilli and qualitative and quantitative blood studies made before the infection, at weekly intervals after the infection and almost daily before and after the administration of tuberculin. Upon completion of the experiment the animals were sacrificed and examined. Tuberculous lesions were found in all the infected animals, but none were present in the controls. Likewise, the blood changes noted in the infected animals were not found in the controls. Following infection, leucocyte counts, while varying considerably, were higher with absolute increases in the lymphocytes and slight increases in the monocytes. Occasional metamyelocytes were seen. The anaemia reported as occurring in tuberculous cattle was noted. Following the first intracutaneous injection of tuberculin an increase in the number of leucocytes was found within twenty-four to seventy-two hours, which consisted primarily of an increase in neutrophiles accompanied by a slight decrease in lymphocytes. Some fluctuation was observed in the controls but there was no disproportional increase of myeloid cells. Twenty-four to forty-eight hours after the

second injection of tuberculin (2 mg.), which was given seven days after the first, more changes in the blood picture were noted. There was a marked absolute increase in neutrophiles with a shift to the left. Promyelocytes, myelocytes and metamyelocytes were easily found. All the types of polymorphonuclears were present. An increase in the monocytes was seen, but it was not as great as that found in the polymorphonuclears. Lymphocytes diminished in number whereas there had been a definite increase in infection and abnormal cells such as the Türck and plasma cells were frequently seen. Despite the absence of the sudden excessive production of neutrophiles seen in rabbits, apparently due to differences in the propensities of the myeloid tissue, it is felt that tuberculin administered to sensitized calves produces a leucocytic reaction comparable quantitatively to that which has been described previously in sensitized rabbits and that the character of the "leukemoid" reaction resembles the condition in human beings which Krumbhaar designated by this term.—*The Character of the Leucocytic Response to Tuberculin in Sensitized Calves, J. Stasney & W. H. Feldman, Am. J. M. Sc., January, 1938, cxcv, 32.*—(*G. F. M.*)

Key to Abstractors

A. A. E.: Adrian A. Ehler, Ann Arbor, Michigan.
A. B. C.: Adele B. Cohn, New York City.
A. P.: Andrew Peters, Springfield, Massachusetts.
C. L. D.: Charles L. Dunham, Chicago, Illinois.
F. B. S.: Florence B. Seibert, Philadelphia, Pennsylvania.
F. G. K.: F. G. Kautz, New York City.
G. F. M.: Gertrude F. Mitchell, Detroit, Michigan.
G. L. L.: George L. Leslie, Howell, Michigan.
G. W. W.: George W. Weber, Albany, New York.
H. J. H.: H. J. Henderson, Philadelphia, Pennsylvania.
H. R. N.: H. R. Nayer, New York City.
H. W. H.: Harlon W. Harrison, Northville, Michigan.
J. E. F.: Jason E. Farber, New York City.
J. G.: J. Goldberg, New York City.
J. S. W.: J. Stanley Woolley, Liberty, New York.
L. F. B.: Lauren F. Busby, Northville, Michigan.
M. B.: Miriam Brailey, Baltimore, Maryland.
M. T. W.: Mildred T. Woolley, Ann. Arbor, Michigan.

INDEX OF ABSTRACTS OF TUBERCULOSIS

Abdominal lymph nodes, Tuberculous disease of, 68
Abscess, Lung, 9
Adenitis, cervical, Tuberculous, 69
Adenoids, tonsils and, tuberculosis of, Prognosis in, 68
Adults, young, children and, in Cattaraugus County, Tuberculosis among, 51
——, ——, in England, Prevention of tuberculosis among, 36
Agassiz, C. D. S. Pneumothorax in children, 53
Age groups, different, Course and mortality of tuberculosis in, 40
Aged, Tuberculin allergy in the, 47
Allergy, Tuberculin, in the aged, 47
Alveolar walls of cat, 77
Amberson, J. B., Jr. Early pulmonary tuberculosis, 55
Anderson, R. S., and Leslie, G. L. Collapse measures, 3
——, ——. ——., —— ——, ——. ——. Collapse therapy results, 4
Anthracosilicosis, Carcinoma in, 19
Apgar, Virginia. See Humphreys, G. H., *et al.*, 84
Aschoff bodies in tuberculous individuals, 34
Aspects of the tuberculosis problem, 47
Atelectasis, 27
—— in children, 52
Atypical spontaneous pneumothorax, 13
Azygos lobe, 76

Bacanu, C. Azygos lobe, 76
Baker-Bates, E. T., and McGibbon, J. E. G. Bronchoscopic investigation of haemoptysis of uncertain cause, 26
Baldwin, Janet, and Thelander, H. E. Variability of findings in tuberculous meningitis, 72
Baylor, J. W., and Bordley, J., III. Prognosis in tuberculosis of tonsils and adenoids, 68
Belgium, rural, Incidence of tuberculous infection in school children in, 40
Belgorod, S. H. Progressive primary complex, 76
Benign tumors of bronchi, 17
Binet, L., and Burstein, M. Nitrogen metabolism in lung, 86
Bliss, T. L. Bronchorrhoea, 8
Block, M., and Rosenblüth, M. B. Pneumococcus pneumonia, 12
Bobrowitz, I. D., and Leon, J. L. Readmissions to a tuberculosis hospital, 63
Boeck's sarcoid, 80
Bogen, E., and Skillen, Jane. Pregnancy and tuberculosis, 63
Bone graft, Short, for spinal fusion, 76
—— marrow changes in tuberculosis, 77
—— tuberculosis, 73
Bones, Multiple cystic tuberculosis of, in children, 55
Bordley, J., III, and Baylor, J. W. Prognosis in tuberculosis of tonsils and adenoids, 68
Böss, C. Mediastinal cysts, 33
Bovine infection, 62
—— tuberculosis in humans in Great Britain, 46
Boynton, Ruth E. See Myers, J. A., *et al.*, 45
Brain, Calcified tuberculoma of, 71
Bronchi, Benign tumors of, 17
——, large, Stenosis of, in pulmonary tuberculosis, 62
Bronchial carcinoma, 18
Bronchiectases, Cystic, 8
Bronchitis, Tuberculous, 63
Bronchogenic distribution of fluid and particulate matter, 86
Bronchomoniliasis, 6
Bronchorrhoea, 8
Bronchoscopic investigation of haemoptysis of uncertain cause, 26

Bronchoscopy in tracheobronchial tuberculosis, 64
Budelmann, G. Vital capacity, 81
Bulla, ruptured, of lung, Fatal spontaneous pneumothorax due to, 13
Bullowa, J. G. M., and Greenbaum, Evelyn. Pneumococcus type-VII pneumonia, 12
Bumbalo, T. S., and Jetter, W. W. Vitamin C in tuberculosis, 55
——, ——. ——., —— ——, ——. ——. Vitamin C in tuberculosis in children, 54
Burgin, L. B., and Higgins, H. L. Phlyctenulosis, 71
Burke, R. M. Vanishing lungs, 24
Burstein, M., and Binet L. Nitrogen metabolism in lung, 86

Calcified tuberculoma of brain, 71
Calves, sensitized, Leucocytic response in, 88
Campbell, J. A. Oxygen administration with box mask and face tent, 35
Canadian hospital workers, Tuberculosis in, 45
Cannetti, G., and Saenz, A. Spontaneous tuberculosis of guinea pigs, 80
Carcinoma, Bronchial, 18
—— in anthracosilicosis, 19
Cardiac failure, Interlobar shadows in, 30
—— output, 84
Cardiologic departments in tuberculosis hospitals, 35
Cardiopulmonary function test, 83
Castex, M. R., and Mazzei, E. S. Atypical spontaneous pneumothorax, 13
——, ——. ——., —— ——, ——. ——. Recurrent benign spontaneous pneumothorax, 15
——, ——. ——., —— ——, ——. ——. Spontaneous benign pneumothorax, 14
Cat, Alveolar walls of, 77
Cattaraugus County, Tuberculosis among children and young adults in, 51
Caud, S. Circulation time, 82
Cavities, closure of, Fat transplant for, 6
——, pulmonary, Encapsulation of, 58
——, Surgical closure of, 5
——, Tuberculous, 57
——, ——, Giant, 57
Cavity, Tuberculous, in infant of eight weeks, 53
Cerebral tuberculosis simulating tumor, 71
Cervical adenitis, Tuberculous, 69
Cervicitis, Tuberculous, 70
Changes in lungs following irradiation, 25
Charr, R. Carcinoma in anthracosilicosis, 19
Chest diseases, Coal miners', in Scotland, 20
——, shape of, Tuberculin reaction and, 54
Children and young adults in Cattaraugus County, Tuberculosis among, 51
——, Atelectasis in, 52
——, Multiple cystic tuberculosis of bones in, 55
——, Pneumothorax in, 53
——, pulmonary fibrosis in, Follow-up of, 27
——, school, in rural Belgium, Incidence of tuberculous infection in, 40
——, Tuberculin reactions in, 47
——, Tuberculosis in, 50, 51
——, —— ——, Vitamin C in, 54
——, Tuberculous peritonitis in, 65
——, ——, Vitamin C in, 55
—— with pulmonary tuberculosis, Results of collapse therapy in, 53
Christian, H. A. Haemothorax in cirrhosis of liver, 35
Christie, R. V. Dyspnoea, 85
Chronic miliary tuberculosis, 56
Circulation, pulmonary, Dual, 85
—— time, 82
——, volume and, of lung, Variations in, 83
Cirrhosis of liver, Haemothorax in, 35
Clark, E., and Rubenfeld, S. Hodgkin's disease of the lungs, 27
Clark, G. M., and Colt, G. H. Tuberculous disease of abdominal lymph nodes, 68
Clerf, L. H. Bronchial carcinoma, 18
Coal miners' chest diseases in Scotland, 20
—— ——, Pneumonoconiosis and tuberculosis in, 19
Collapse measures, 3
—— therapy in children with pulmonary tuberculosis, Results of, 53
—— —— results, 4
Colt, G. H., and Clark, G. M. Tuberculous disease of abdominal lymph nodes, 68
Complex, primary, Exacerbation of, 53
——, ——, Progressive, 76
Congenital cystic disease of lung, 22
—— —— ——, Total pneumonectomy for, 22
—— cysts of lung, 23

Control of tuberculosis, 49
Coon, H. M. See Gale, J. W., *et al.*, 22
Cooper, D. A., and Erb, W. H. Death following phrenicectomy, 2
Corper, H. J. Tuberculous infection in guinea pigs, 80
Coryllos, P. N. Tuberculous cavities, 57
——, ——. ——., and Hochberg, L. A. Thoracoplasty with pneumothorax and pleural effusion, 2
——, ——. ——., —— Ornstein, G. G. Giant tuberculous cavities, 57
——, ——. ——., —— Weinstein, M. Subtotal scapulectomy, 6
Coughing, Mechanism of, 86
Course and mortality of tuberculosis in different age groups, 40
Courville, C. B., and Evans, H. S. Calcified tuberculoma of brain, 71
Croizier, and Martin, E. Pulmonary fibrosis in miners, 19
Cystic bronchiectases, 8
—— disease, Congenital, of lung, 22
—— ——, ——, Total pneumonectomy for, 22
—— tuberculosis, Multiple, of bones in children, 55
Cysts, Congenital, of lung, 23
——, Lung, 23
——, Mediastinal, 33

Dauer, C. C. Trends of tuberculosis mortality by sex, 41
Death following phrenicectomy, 2
Decline in tuberculosis mortality, 42
Derscheid, G., and Toussaint, P. Encapsulation of pulmonary cavities, 58
Detroit, Tuberculosis prevention in, 48
Diabetes and tuberculosis, 61
Diabetic coma, Tuberculous meningitis resembling, 72
Diaphragmatic hernia, 31
—— ——, Traumatic, 32
Diehl, H. S. See Myers, J. A., *et al.*, 45
Dirkse, P. R., and Peirce, C. B. Pulmonary pneumatocele, 22
Diseases, chest, Coal miners', in Scotland, 20
Distribution, Bronchogenic, of fluid and particulate matter, 86
Dual pulmonary circulation, 85
Duodenum, oesophagus, stomach and, Pressures in, 86
Durand, H. Lung abscess, 9
Dye, Spread of, in skin of tuberculous guinea pigs, 88
Dyspnoea, 85

Early pulmonary tuberculosis, 55
Effusion, Interlobar, 31
——, pleural, pneumothorax and, Thoracoplasty with, 2
Ellison, R. T. Mediastinal hernia, 32
Empyema, Periapical, 29
——, Tuberculous, 64
Encapsulation of pulmonary cavities, 58
England, Prevention of tuberculosis among young adults in, 36
Englebreth-Holm, J. Tuberculosis splenomegaly, 67
Epituberculosis, 52
Epstein, I. G., and Ornstein, G. G. Tuberculous bronchitis, 63
Erb, W. H., and Cooper, D. A. Death following phrenicectomy, 2
Erythema nodosum and pulmonary tuberculosis, 52
Evans, H. S., and Courville, C. B. Calcified tuberculoma of brain, 71
Exacerbation of primary complex, 53
Experimental tuberculous panophthalmitis, 87
Extrathoracic, Intra- and, tuberculosis, 76

Fat transplant for closure of cavities, 6
Fatal haemorrhage, 76
—— spontaneous pneumothorax due to ruptured bulla of lung, 13
Feldman, W. H., and Stasney, J. Leucocytic response in sensitized calves, 88
Ferrando, G., and Rabino, A. Social importance of the modern sanatorium in Italy, 44
Fetter, W. Epituberculosis, 52
Fibrosis, pulmonary, in children, Follow-up of, 27
——, ——, —— miners, 19
Fineman, S. Jejunum in tuberculosis, 66
Fixation, Spinal, 75
Fluid and particulate matter, Bronchogenic distribution of, 86

Follow-up of pulmonary fibrosis in children, 27
Fractures of ribs, Spontaneous, in pulmonary tuberculosis, 62
Frimodt-Möller, C. Tuberculosis problem in India, 49
Frost, W. H. Control of tuberculosis, 49
Function, Pulmonary, 81
Funschtein, L. Hodgkin's disease and tuberculosis, 27
Fusion, Spinal, in tuberculosis of spine, 75
——, ——, Short bone graft for, 76

Gale, J. W., Kealey, J. L., and Coon, H. M. Total pneumonectomy for congenital cystic disease, 22
Gaucher's disease of lungs, 27
Giant tuberculous cavities, 57
Gilbert, Lilian. See Lincoln, Edith M., *et al.*, 47
Goldberger, Esther. See Kereszturi, Camille, *et al.*, 47
Gough, J. Fatal spontaneous pneumothorax due to ruptured bulla of lung, 13
Graham, E. A. See Tuttle, W. M., *et al.*, 1
Gray, W. A. Experimental tuberculous panophthalmitis, 87
Great Britain and South Africa, Silicosis laws in, 21
—— ——, Bovine tuberculosis in humans in, 46
Green, H. Exacerbation of primary complex, 53
Greenbaum, Evelyn, and Bullowa, J. G. M. Pneumococcus type-VII pneumonia, 12
Greger, C. Skin temperatures in tuberculosis of joints, 73
Grevle, A. Hospital observation on spondylitic patients, 75
Griffith, A. S. Bovine tuberculosis in humans in Great Britain, 46
Guinea pigs, Spontaneous tuberculosis of, 80
—— ——, Tuberculous infection in, 80
—— ——, ——, Spread of dye in skin of, 88

Haberland, H. O. F. Bone tuberculosis, 73
Haematogenous pulmonary tuberculosis, 56
Haemopneumothorax, Spontaneous, 16
Haemoptysis, 57
—— of uncertain cause, Bronchoscopic investigation of, 26
Haemorrhage, Fatal, 76
Haemothorax in cirrhosis of liver, 35
Hall, J. A. M. Coal miners' chest diseases in Scotland, 20
Hamada, G. Spinal fixation, 75
Hamperl, H. Benign tumors of bronchi, 17
Harmon, G. E. See Vaughn, H. F., *et al.*, 48
Hart, P. M. D'A. Prevention of tuberculosis among young adults in England, 36
Hatcher, C. H., and Phemister, D. B. Tuberculous infection of the hip joint, 73
Hausbrandt, F. Intimal changes in branches of portal vein, 78
Heart in pulmonary tuberculosis, 62
Hellstadius, A. Tuberculous spondylitis, 75
Hernia, Diaphragmatic, 31
——, ——, Traumatic, 32
——, Mediastinal, 32
Hertzberg, G. Tuberculosis in two Norwegian forest districts, 42
Higgins, H. L., and Burgin, L. B. Phlyctenulosis, 71
Himsworth, H. P. Diabetes and tuberculosis, 61
Hip joint, Tuberculous infection of the, 73
Hochberg, L. A., and Coryllos, P. N. Thoracoplasty with pneumothorax and pleural effusion, 2
Hodgkin's disease and tuberculosis, 27
—— —— of the lungs, 27
Holman, E. Partial resection of lower scapula, 5
Hopkins, H. U. Spontaneous haemopneumothrax, 16
Hospital observation on spondylitic patients, 75
—— personnel, Tuberculosis in, 45
——, tuberculosis, Readmissions to a, 63
—— workers, Canadian, Tuberculosis in, 45
Hospitals, tuberculosis, Cardiologic departments in, 35
Households, Periodic accrediting of, 36
Hoyle, C. Chronic miliary tuberculosis, 56
Hsieh, C. K., and Kimm, H. T. Changes in lungs following irradiation, 25
Humphreys, G. H., Moore, R. L., Maier, H. C., and Apgar, Virginia. Cardiac output, 84
Hypersensitiveness, Tuberculin, 1

Ikeda, K. Bronchomoniliasis, 6
Immunity, Scrofula and, in tuberculosis, 70
Incidence of tuberculous infection in school children in rural Belgium, 40
India, Tuberculosis problem in, 49
Industrial workers, Pneumonia and tuberculosis among, 46
Industry, Tuberculosis problem in, 43
Infant of eight weeks, Tuberculous cavity in, 53
Infants, Tuberculosis in, 50
Infarction of lung, 26
Infection, Bovine, 62
——, Tuberculous, among soldiers, 41
——, ——, in guinea pigs, 80
——, ——, Incidence of, in school chidren in rural Belgium, 40
——, ——, of the hip joint, 73
Interlobar effusion, 31
—— shadows in cardiac failure, 30
Intestinal tuberculosis, 66
Intimal changes in branches of portal vein, 78
Intra- and extrathoracic tuberculosis, 76
Irradiation, Changes in lungs following, 25
Ischium, Tuberculous osteitis of the, 73
Italy, Social importance of the modern sanatorium in, 44
——, Tuberculosis morbidity in, 41

Jejunum in tuberculosis, 66
Jetter, W. W , and Bumbalo, T. S. Vitamin C in tuberculosis, 55
——, ——. ——., —— ——, ——. ——. Vitamin C in tuberculosis in children, 54
Johnstone, J. G. Treatment of joint tuberculosis, 72
Joint, hip, Tuberculous infection of the, 73
—— tuberculosis, Treatment of, 72
Joints, tuberculosis of, Skin temperatures in, 73
Jonnesco, D., and Stoïchitza, N. N. Pulmonary mycosis, 7
Joyner, A. L., and Sabin, F. R. Spread of dye in skin of tuberculous guinea pigs, 88

Kaunitz, J. Atelectasis, 27
Kautz, F. G., and Pinner, M. Periapical empyema, 29
Kealey, J. L. See Gale, J. W., *et al.*, 22
Kereszturi, Camille, Goldberger, Esther, and Nojima, Kimi. Tuberculin allergy in the aged, 47
Kibbey, C. H. Pneumonia and tuberculosis among industrial workers, 46
Kimm, H. T., and Hsieh, C. K. Changes in lungs following irradiation, 25
Klosk, E. Fatal haemorrhage, 76
Kornat, M. Tuberculosis in children, 51
Korns, J. H. Tuberculosis among children and young adults in Cattaraugus County, 51
Krafchik, L. L., and Slobody, L. B. Tuberculous meningitis resembling diabetic coma, 72

Lane, R. E. Tuberculosis problem in industry, 43
Lanza, G. Bone marrow changes in tuberculosis, 77
Law, J. L., and Perham, W. S. Multiple cystic tuberculosis of bones in children, 55
Leibovici, D., and Ornstein, G. G. Pressures in oesophagus, stomach and duodenum, 86
Leon, J. L., and Bobrowitz, I. D. Readmissions to a tuberculosis hospital, 63
Leslie, G. L., and Anderson, R. S. Collapse measures, 3
——, ——. ——., —— ——, ——. ——. Collapse therapy results, 4
Leucocytic response in sensitized calves, 88
Leverton, W. R. Heart in pulmonary tuberculosis, 62
Levine, H. B., and White, P. D. Infarction of lung, 26
Levitin, J. Interlobar effusion, 31
Lincoln, Edith M., Raia, Antoinette, and Gilbert, Lilian. Tuberculin reactions in children, 47
Liver, cirrhosis of, Haemothorax in, 35
Lung abscess, 9
——, Congenital cystic disease of, 22
——, —— cysts of, 23
—— cysts, 23
—— in xanthomatosis, 28
——, Infarction of, 26
——, Nitrogen metabolism in, 86
——, ruptured bulla of, Fatal spontaneous pneumothorax due to, 13

Lung, tuberculosis of, Vascular changes in, 78
——, volume and circulation of, Variations in, 83
Lungs, Changes in, following irradiation, 25
——, Gaucher's disease of, 27
——, Hodgkin's disease of the, 27
——, Vanishing, 24
Lymph nodes, abdominal, Tuberculous disease of, 68

MacIntyre, I. C. Tuberculosis in New Zealand, 42
Macklin, C. C. Alveolar walls of cat, 77
Macklin, Madge T. Tuberculosis in Canadian hospital workers, 45
Magnusson, R. Tuberculous osteitis of the ischium, 73
Magnússon, S. Course and mortality of tuberculosis in different age groups, 40
Maier, H. C. See Humphreys, G. H., *et al.*, 84
Marfan, A. B. Scrofula and immunity in tuberculosis, 70
Marks. J. H. Diaphragmatic hernia, 31
Martin, E., and Croizier. Pulmonary fibrosis in miners, 19
Mask, box, and face tent, Oxygen administration with, 35
Mast, W. H., and McDonough, J. P. Traumatic diaphragmatic hernia, 32
Masugi, M., Murasawa, S., and Yä, S. Aschoff bodies in tuberculous individuals, 34
Mazzei, E. S., and Castex, M. R. Atypical spontaneous pneumothorax, 13
——, ——. ——., —— ——, ——. ——. Recurrent benign spontaneous pneumothorax, 15
——, ——. ——., —— ——, ——. ——. Spontaneous benign pneumothorax, 14
McDonough, J. P., and Mast, W. H. Traumatic diaphragmatic hernia, 32
McGibbon, J. E. G., and Baker-Bates, E. T. Bronchoscopic investigation of haemoptysis of uncertain cause, 26
Mechanism of coughing, 86
Mediastinal cysts, 33
—— hernia, 32
Meersseman, F. Tuberculous infection among soldiers, 41
Meningitis, Tuberculous, resembling diabetic coma, 72
——, ——, Variability of findings in, 72
Metabolism, Nitrogen, in lung, 86
Miliary tuberculosis, Chronic, 56
Miller, J. A. Unsolved problems of tuberculosis, 39
——, ——. ——., and Rappaport, I. Pulmonary function, 81
Miller, Miriam, and Wood, D. A. Dual pulmonary circulation, 85
Miners', Coal, chest diseases in Scotland, 20
——, ——, Pneumonoconiosis and tuberculosis in, 19
——, Pulmonary fibrosis in, 19
Mitchell, Gertrude F. Tuberculous pericarditis and Pick's disease, 66
Molner, J. G. See Vaughn, H. F., *et al.*, 48
Moore, G. A. Intestinal tuberculosis, 66
Moore, R. L. See Humphreys, G. H., *et al.*, 84
Morbidity, Tuberculosis, in Italy, 41
Mortality of tuberculosis in different age groups, Course and, 40
——, tuberculosis, Decline in, 42
——, ——, Trends of, by sex, 41
Moskacheva, K. A., and Reinberg, S. A. Lung in xanthomatosis, 28
Müller, E. M. Diabetes and tuberculosis, 61
Multiple cystic tuberculosis of bones in children, 55
Murasawa, S. See Masugi, M., *et al.*, 34
Mycosis, Pulmonary, 7
Myers, B. Gaucher's disease of lungs, 27
Myers, J. A., Trach, B., Diehl, H. S., and Boynton, Ruth E. Tuberculosis in hospital personnel, 45
Myerson, M. C. Bronchoscopy in tracheobronchial tuberculosis, 64

Nayer, H. R. Intestinal tuberculosis, 66
Neubert, B. Vascular changes in tuberculosis of lung, 78
Neuhoff, H. Fat transplant for closure of cavities, 6
New Zealand, Tuberculosis in, 42
Nitrogen metabolism in lung, 86
Nojima, Kimi. See Kereszturi, Camille, *et al.*, 47
Norwegian forest districts, two, Tuberculosis in, 42

Nüss, M. Bovine infection, 62
Nylin, G. Cardiopulmonary function test, 83

O'Brien, E. J. See Tuttle, W. M., *et al.*, 1
Observation, Hospital, on spondylitic patients, 75
Oesophagus, stomach and duodenum, Pressures in, 86
Ornstein, G. G., and Coryllos, P. N. Giant tuberculous cavities, 57
——, ——. ——., —— Epstein, I. G. Tuberculous bronchitis, 63
——, ——. ——., —— Leibovici, D. Pressures in oesophagus, stomach and duodenum, 86
Ortega, L., and Verdes. Cardiologic departments in tuberculosis hospitals, 35
Osteitis, Tuberculous, of the ischium, 73
Oxygen administration with box mask and face tent, 35

Panophthalmitis, tuberculous, Experimental, 87
Parodi, F. Variations in volume and circulation of lung, 83
Partial resection of lower scapula, 5
Particulate matter, fluid and, Bronchogenic distribution of, 86
Peirce, C. B., and Dirkse, P. R. Pulmonary pneumatocele, 22
Perham, W. S., and Law, J. L. Multiple cystic tuberculosis of bones in children, 55
Periapical empyema, 29
Pericarditis, Tuberculous, and Pick's disease, 66
Periodic accrediting of households, 36
Peritonitis, Tuberculous, in children, 65
Petter, C. K. Intra- and extrathoracic tuberculosis, 76
Phemister, D. B., and Hatcher, C. H. Tuberculous infection of the hip joint, 73
Phlyctenulosis, 71
Phrenicectomy, Death following, 2
Pick's disease, Tuberculous pericarditis and, 66
Pinner, M., and Kautz, F. G. Periapical empyema, 29
Pleural effusion, pneumothorax and, Thoracoplasty with, 2
Pneumatocele, Pulmonary, 22
Pneumococcus pneumonia, 12
—— type-VII pneumonia, 12
Pneumonectomy, Total, for congenital cystic disease, 22
Pneumonia and tuberculosis among industrial workers, 46
——, Pneumococcus, 12
——, —— type-VII, 12
Pneumonoconiosis and tuberculosis in coal miners, 19
Pneumothorax and pleural effusion, Thoracoplasty with, 2
——, benign, Spontaneous, 14
—— in children, 53
——, spontaneous, Atypical, 13
——, ——, benign, Recurrent, 15
——, ——, Fatal, due to ruptured bulla of lung, 13
Portal vein, Intimal changes in branches of, 78
Pregnancy and tuberculosis, 63
Pressures in oesophagus, stomach and duodenum, 86
Prevention of tuberculosis among young adults in England, 36
——, Tuberculosis, in Detroit, 48
Price, D. Tuberculosis in infants, 50
——, ——. Tuberculous cavity in infant of eight weeks, 53
Primary complex, Exacerbation of, 53
—— ——, Progressive, 76
Prognosis in tuberculosis of tonsils and adenoids, 68
Progressive primary complex, 76
Pseudocavitation, 25
Pulmonary cavities, Encapsulation of, 58
—— circulation, Dual, 85
—— fibrosis in children, Follow-up of, 27
—— —— —— miners, 19
—— function, 81
—— mycosis, 7
—— pneumatocele, 22
—— tuberculosis, children with, Results of collapse therapy in, 53
—— ——, Early, 55
—— ——, Erythema nodosum and, 52
—— ——, Haematogenous, 56
—— ——, Heart in, 62
—— ——, Spontaneous fractures of ribs in, 62
—— ——, Stenosis of large bronchi in, 62

Rabino, A., and Ferrando, G. Social importance of the modern sanatorium in Italy, 44
Rabinowitz, L., and Rogers, E. J. Lung cysts, 23
Raia, Antoinette. See Lincoln, Edith M., *et al.*, 47
Rappaport, I., and Miller, J. A. Pulmonary function, 81
Raven, M. O. Follow-up of pulmonary fibrosis in children, 27
Reaction, Tuberculin, and shape of chest, 54
Reactions, Tuberculin, in children, 47
Readmissions to a tuberculosis hospital, 63
Recurrent benign spontaneous pneumothorax, 15
Reichle, H. S. Bronchogenic distribution of fluid and particulate matter, 86
Reid, B., and Wilkinson, M. C. Tuberculous cervical adenitis, 69
Reinberg, S. A., and Moskacheva, K. A. Lung in xanthomatosis, 28
Reisner, D., and Tchertkoff, I. G. Cystic bronchiectases, 8
Resection, Partial, of lower scapula, 5
Results, Collapse therapy, 4
—— of collapse therapy in children with pulmonary tuberculosis, 53
Ribs, Spontaneous fractures of, in pulmonary tuberculosis, 62
Rogers, E. J., and Rabinowitz, L. Lung cysts, 23
Rosenblüth, M. B., and Block, M. Pneumococcus pneumonia, 12
Roubier, C. Interlobar shadows in cardiac failure, 30
Rubenfeld, S., and Clark, E. Hodgkin's disease of the lungs, 27
Rural Belgium, Incidence of tuberculous infection in school children in, 40
Rykels, D. K. Decline in tuberculosis mortality, 42

Sabbione, C. Spontaneous fractures of ribs in pulmonary tuberculosis, 62
Sabin, F. R., and Joyner, A. L. Spread of dye in skin of tuberculous guinea pigs, 88
Sacro-iliac tuberculosis, 74
Saenz, A., and Cannetti, G. Spontaneous tuberculosis of guinea pigs, 80
Sanatorium, modern, Social importance of the, in Italy, 44
Sandler, E. Stenosis of large bronchi in pulmonary tuberculosis, 62
Scapula, lower, Partial resection of, 5
Scapulectomy, Subtotal, 6
Schenck, S. G. Congenital cysts of lung, 23
Schumann, C. Pseudocavitation, 25
Scotland, Coal miners' chest diseases in, 20
Scrofula and immunity in tuberculosis, 70
Sellors, T. H. Surgical closure of cavities, 5
Sen, P. K. Pneumonoconiosis and tuberculosis in coal miners, 19
Sensitized calves, Leucocytic response in, 88
Sex, Trends of tuberculosis mortality by, 41
Shadows, Interlobar, in cardiac failure, 30
Short bone graft for spinal fusion, 76
Siegal, M., and Singer, B. Results of collapse therapy in children with pulmonary tuberculosis, 53
Silicosis laws in Great Britain and South Africa, 21
Singer, B., and Siegal, M. Results of collapse therapy in children with pulmonary tuberculosis, 53
——, ——., —— Van Bark, B. Vitamin C in tuberculous children, 55
Skillen, Jane, and Bogen, E. Pregnancy and tuberculosis, 63
Skin of tuberculous guinea pigs, Spread of dye in, 88
—— temperatures in tuberculosis of joints, 73
Slobody, L. B., and Krafchik, L. L. Tuberculous meningitis resembling diabetic coma, 72
Social importance of the modern sanatorium in Italy, 44
Soldiers, Tuberculous infection among, 41
South Africa, Great Britain and, Silicosis laws in, 21
Spencer, J., and Warren, S. Boeck's sarcoid, 80
Spinal fixation, 75
—— fusion in tuberculosis of spine, 75
—— ——, Short bone graft for, 76
Spine, tuberculosis of, Spinal fusion in, 75
Splenomegaly, Tuberculosis, 67
Spondylitic patients, Hospital observation on, 75

Spondylitis, Tuberculous, 75
Spontaneous benign pneumothorax, 14
—— fractures of ribs in pulmonary tuberculosis, 62
—— haemopneumothorax, 16
—— pneumothorax, Atypical, 13
—— ——, benign, Recurrent, 15
—— ——, Fatal, due to ruptured bulla of lung, 13
—— tuberculosis of guinea pigs, 80
Spread of dye in skin of tuberculous guinea pigs, 88
Stasney, J., and Feldman, W. H. Leucocytic response in sensitized calves, 88
Stenosis of large bronchi in pulmonary tuberculosis, 62
Stevenson, C. S. Tuberculous cervicitis, 70
Stewart, C. A. Periodic accrediting of households, 36
Stoichitza, N. N., and Jonnesco, D. Pulmonary mycosis, 7
Stomach, oesophagus, and duodenum, Pressures in, 86
Subtotal scapulectomy, 6
Surgical closure of cavities, 5
Sutherland, D. P. Aspects of the tuberculosis problem, 47
Swift, W. E. Spinal fusion in tuberculosis of spine, 75

Tchertkoff, I. G., and Reisner, D. Cystic bronchiectases, 8
Temperatures, Skin, in tuberculosis of joints, 73
Tent, face, box mask and, Oxygen administration with, 35
Test, Cardiopulmonary function, 83
Thelander, H. E., and Baldwin, Janet. Variability of findings in tuberculous meningitis, 72
Therapy, collapse, in children with pulmonary tuberculosis, Results of, 53
——, ——, results, 4
Thompson, F. Sacro-iliac tuberculosis, 74
Thoracoplasty with pneumothorax and pleural effusion, 2
Tonsils and adenoids, tuberculosis of, Prognosis in, 68
Total pneumonectomy for congenital cystic disease, 22
Toussaint, P., and Derscheid, G. Encapsulation of pulmonary cavities, 58
Trach, B. See Myers, J. A., *et al.*, 45
Tracheobronchial tuberculosis, Bronchoscopy in, 64
Transplant, Fat, for closure of cavities, 6
Traumatic diaphragmatic hernia, 32
Treatment of joint tuberculosis, 72
Trends of tuberculosis mortality by sex, 41
Tuberculin allergy in the aged, 47
—— hypersensitiveness, 1
—— reaction and shape of chest, 54
—— reactions in children, 47
Tuberculoma of brain, Calcified, 71
Tuberculosis among children and young adults in Cattaraugus County, 51
——, Bone, 73
——, —— marrow changes in, 77
——, Bovine, in humans in Great Britain, 46
——, Cerebral, simulating tumor, 71
——, Control of, 49
——, cystic, Multiple, of bones in children, 55
——, Diabetes and, 61
——, Hodgkin's disease and, 27
—— hospital, Readmissions to a, 63
—— hospitals, Cardiologic departments in, 35
—— in Canadian hospital workers, 45
—— —— children, 50, 51
—— —— ——, Vitamin C in, 54
—— —— coal miners, Pneumonoconiosis and, 19
—— —— different age groups, Course and mortality of, 40
—— —— hospital personnel, 45
—— —— infants, 50
—— —— New Zealand, 42
—— —— two Norwegian forest districts, 42
——, Intestinal, 66
——, Intra- and extrathoracic, 76
——, Jejunum in, 66
——, joint, Treatment of, 72
——, miliary, Chronic, 56
—— morbidity in Italy, 41
—— mortality, Decline in, 42
—— ——, Trends of, by sex, 41
—— of joints, Skin temperatures in, 73
—— —— lung, Vascular changes in, 78
—— —— spine, Spinal fusion in, 75

Tuberculosis of tonsils and adenoids, Prognosis in, 68
——, Pneumonia and, among industrial workers, 46
——, Pregnancy and, 63
—— prevention in Detroit, 48
——, —— of, among young adults in England, 36
—— problem, Aspects of the, 47
—— —— in India, 49
—— —— —— industry, 43
——, pulmonary, children with, Results of collapse therapy in, 53
——, ——, Early, 55
——, ——, Erythema nodosum and, 52
——, ——, Haematogenous, 56
——, ——, Heart in, 62
——, ——, Spontaneous fractures of ribs in, 62
——, ——, Stenosis of large bronchi in, 62
——, Sacro-iliac, 74
——, Scrofula and immunity in, 70
—— splenomegaly, 67
——, Spontaneous, of guinea pigs, 80
——, tracheobronchial, Bronchoscopy in, 64
——, Unsolved problems of, 39
——, Vitamin C in, 55
Tuberculous bronchitis, 63
—— cavities, 57
—— ——, Giant, 57
—— cavity in infant of eight weeks, 53
—— cervical adenitis, 69
—— cervicitis, 70
—— children, Vitamin C in, 55
—— disease of abdominal lymph nodes, 68
—— empyema, 64
—— guinea pigs, Spread of dye in skin of, 88
—— individuals, Aschoff bodies in, 34
—— infection among soldiers, 41
—— —— in guinea pigs, 80
—— ——, Incidence of, in school children in rural Belgium, 40
—— —— of the hip joint, 73
—— meningitis resembling diabetic coma, 72
—— ——, Variability of findings in, 72
—— osteitis of the ischium, 73
—— panophthalmitis, Experimental, 87
—— pericarditis and Pick's disease, 66
—— peritonitis in children, 65
—— spondylitis, 75
Tumor, Cerebral tuberculosis simulating, 71
Tumors, Benign, of bronchi, 17
Tuttle, W. M., O'Brien, E. J., and Graham, E. A. Tuberculin hypersensitiveness, 1

Unsolved problems of tuberculosis, 39
Urechia, C. Cerebral tuberculosis simulating tumor, 71

Van Antwerp, L. D. Tuberculous peritonitis in children, 65
Van Bark, B., and Singer, B. Vitamin C in tuberculous children, 55
van den Eeckhout, H. Incidence of tuberculous infection in school children in rural Belgium, 40
Van Otterloo, J. De Mol. Short bone graft for spinal fusion, 76
Vanishing lungs, 24
Variability of findings in tuberculous meningitis, 72
Variations in volume and circulation of lung, 83
Vascular changes in tuberculosis of lung, 78
Vaughn, H. F., Harmon, G. E., and Molner, J. G. Tuberculosis prevention in Detroit, 48
Vein, portal, Intimal changes in branches of, 78
Verdes, and Ortega, L. Cardiologic departments in tuberculosis hospitals, 35
Viethen, A. Tuberculosis in children, 50
Vital capacity, 81
Vitamin C in tuberculosis, 55
—— —— —— —— in children, 54
—— —— —— tuberculous children, 55
Volume and circulation of lung, Variations in, 83

Wallgren, A. Erythema nodosum and pulmonary tuberculosis, 52
Walter-Regensburg, E. Haemoptysis, 57
Warren, S., and Spencer, J. Boeck's sarcoid, 80
Weber, H. H. Mechanism of coughing, 86
Weinstein, M., and Coryllos, P. N. Subtotal scapulectomy, 6
Weisman, S. A. Tuberculin reaction and shape of chest, 54
White, P. D., and Levine, H. B. Infarction of lung, 26

Wilkinson, M. C., and Reid, B. Tuberculous cervical adenitis, 69
Wood, D. A., and Miller, Miriam. Dual pulmonary circulation, 85
Wood, H. G. Congenital cystic disease of lung, 22
Woodruff, W. Tuberculous empyema, 64
Xanthomatosis, Lung in, 28
Yä, S. See Masugi, M., *et al.*, 34
Zavod, W. A. Haematogenous pulmonary tuberculosis, 56
Zeyland, J. Atelectasis in children, 52